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EXOPHTHALMOS ASSOCIATED WITH DIABETES INSIPIDUS AND LARGE DEFECTS IN THE BONES OF THE SKULL*

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In November, 1927, Charlie Chiarelli, an Italian boy, nearly 4 years old, was admitted to Bellevue Hospital, and was assigned to the eye service because of marked exophthalmos. On seeing him, I recognized him immediately as a patient whom I had examined at Mount Sinai Hospital about three months before, through the courtesy of Dr. Isadore Goldstein. Fortunately, the diagnosis of Schüller's disease (or Christian's disease) had been made at Mount Sinai Hospital before I saw the case, for I was unfamiliar with the striking syndrome presented and should not have been able to classify it. The case in its earlier stages has been reported by Dr. Louis Hausman and Dr. Walter Bromberg in the *Archives of Neurology and Psychiatry*.[1]

Pronounced exophthalmos was the most striking clinical feature in the reported cases, but seemingly the attention of ophthalmologists has not been called to the condition by descriptions in the literature on the eyes. Moreover, no reference can be found to an antemortem orbital dissection in a case demonstrating the syndrome, and so for the first time I can report surgical exploration of the orbit in a living victim of this rare and important disease and definitely establish the cause of the extreme proptosis.

In September, 1905, Dr. Thomas W. Kay[2] presented before the Medical Society of Pennsylvania a 7 year old boy who had had scarlatina three years before. Eighteen months later, exophthalmos had made its appearance and had progressed steadily. The author said that the exophthalmos was so severe that the eyes had to be protected by a handkerchief as they were too prominent for the use of glasses. Four months after the appearance of exophthalmos, polyuria was evidenced. At the time the case was presented, the specific gravity of the urine varied from 1.000 to 1.001, and the child was passing 23 quarts of urine daily. Large parts of the bones of the skull were soft, and the

* Submitted for publication, Sept. 16, 1924.

1. Hausman, Louis, and Bromberg, Walter: Diabetic Exophthalmic Dysostosis, Arch. Neurol. & Psychiat. **21**:1402 (June) 1929.

2. Kay, Thomas W.: Acquired Hydrocephalus with Atrophic Bone Changes, Exophthalmos and Polyuria, Pennsylvania M. J. **9**:520, 1905-1906.

squamous portion of the right temporal bone gave way a little under pressure of the finger. Dr. Kay expressed the opinion that in this case the possible lesion was situated in or about the floor of the fourth ventricle.

In 1915, Prof. Artur Schüller [3] described three cases, in one of which, that of a 5 year old girl, bilateral exophthalmos, large defects in the membranous bones and polyuria were manifested. Schüller was especially interested in the roentgen appearance of the skull, which he described as "maplike," on account of the transparent spots seen in the x-ray pictures. He could palpate pulsating openings. He supposed that the defects in the skull and the diabetes insipidus were caused by disturbance of the pituitary function, and he thought the exophthalmos was occasioned by the pressure of the brain on the orbital contents made possible by the large defects in the anterior fossa of the skull.

In the Osler Memorial Volume of 1919, in an article by Dr. Henry A. Christian,[4] later reprinted in the *Medical Clinics of North America,* is the report of the case of a 5 year old girl, who was seen at Peter Bent Brigham Hospital in Boston. On account of his excellent description of the condition, the syndrome is sometimes called Christian's disease. Christian considered the syndrome essentially one of dyspituitarism. He thought the disturbed pituitary function occasioned both the diabetes insipidus and the changes in the bone. He said, "The most extensive defects are in the anterior half of the skull. Of the frontal bone, particularly the lateral portions, only irregular rather narrow septa of bones are left between the large islands of entire bone disappearance. In a similar way the orbital plates of the frontal bone have largely disappeared so that there is little bony support remaining for the eyeballs." Christian found that pituitary extract controlled the polyuria when given subcutaneously, but not when given by other methods. Pituitary extract had no effect on the bone defects or exophthalmos.

In 1921, Dr. Alfred Hand [5] recalled a report made by him in 1893 [6] in which he described the case of a boy 3 years of age who was admitted to the Children's Hospital in Philadelphia on Dec. 1, 1892. The child's eyes were in a condition of exophthalmos, giving him a froglike appear-

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3. Schüller, Artur: Ueber eigenartige Schadeldefekte in Jugendalter, Fortschr. a. d. Geb. d. Röntgenstrahlen **23**:12, 1915-1916.

4. Christian, H. A.: Defects in Membranous Bones, Exophthalmos and Diabetes Insipidus: An Unusual Syndrome of Dyspituitarism, Contrib. Med. & Biol. Research **1**:390, 1919; M. Clin. North America **3**:847, 1920.

5. Hand, A.: Defects of Membranous Bones, Exophthalmos and Polyuria in Childhood: Is It Dyspituitarism? Am. J. M. Sc. **162**:509, 1921.

6. Hand, A.: Polyuria and Tuberculosis, Arch. Pediat. **10**:673, 1893.

ance, and there was a corneal opacity in each eye. He was suffering from thirst and was passing 114 ounces of urine daily. The specific gravity was 1.000, and the urine contained no sugar or albumin. Bronchopneumonia developed and the child died. From a general view of the case, a diagnosis of tuberculosis was made. At autopsy, a yellow spot about the size of a five cent piece was noticed about the right parietal eminence. After reading the published report of Kay's case and with increased experience at the autopsy table, and with Christian's case report in mind, Hand doubted whether the tuberculosis had any causal relation to the exophthalmos, polyuria or defect with the yellow spot in the parietal bone. He described another case of a 4 year old boy who had exophthalmos and skull defects, but who at that time had no polyuria.[6] Hand adopted Schüller's suggestion that the exophthalmos was due to involvement of the orbital plate of the frontal bone, but also advanced the theory that "the primary process might be neoplastic, benign and myxomatous in character effecting for some unknown reason the membranous bones and producing exophthalmos and polyuria secondarily by pressure."

Thompson, Keegan and Dunn [7] reported the painstaking study of a 9 year old boy who manifested the typical syndrome. The boy died, and the authors were then able to give a detailed account of the autopsy observations, but apparently the orbit was not studied at autopsy. "In the center of the largest membranous areas, there was yellowish fibrous tissue and the inner surface of the dura was mottled by a yellowish tissue. Microscopic section through the region showed a lining layer of large oval cells with sharp borders with clear, slightly granular cytoplasm and small compact centrally placed nuclei. The cytoplasm contained a variable amount of lipoid material in finely divided form." Death was ascribed to cardiac failure attributed to impaired circulation, incident to extensive pulmonary fibrosis. The authors said that "the faulting of the frontal bones with its orbital plates fully explains the exophthalmos." They stressed the importance of early involvement of the tuber cinereum, and dyspituitarism as a cause of the syndrome was excluded by the absence of material pathologic changes in the hypophysis. Pathologic support was given to current work on experimental polyuria which puts the lesion of diabetes insipidus in the hypothalamus. From clinical behavior and pathologic changes, the authors judge that an infectious etiology is more likely than a primary metabolic endocrinologic disturbance.

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7. Thompson, C. O.; Keegan, J. J., and Dunn, A. D.: Defects of Membranous Bones, Exophthalmos and Diabetes Insipidus: Report of a Case with Necropsy, Arch. Int. Med. **36**:650 (Nov.) 1925.

Grosh and Stifel [8] reported a case of a 7 year old girl who came under observation on June 27, 1919, in Toledo. In addition to marked defects in the bones of the skull, diabetes insipidus and a definite exophthalmos of the left eye, there was some degree of dwarfism. The authors called attention to the work of Bremer and Bailey, showing that polyuria found in experimental lesions at the hypophysis is usually temporary, while lesions of the hypothalamus produce a permanent polyuria.

In 1926, Denzer [9] briefly reported the case of a 4½ year old boy who had defects in the skull bones a year before polyuria was discovered. Denzer said that the exophthalmos was probably due to defects in the bones of the orbit.

In 1927, W. R. Stowe [10] reported briefly a case of diabetes insipidus associated with defects in the skull. The clinical picture was diabetes insipidus with exophthalmos. Stowe considered that roentgenograms should be made of the skull in all cases of diabetes insipidus in children. Later, D. S. Milne reported the same case more in detail to R. S. Rowland. An autopsy was performed, and "on reflecting the scalp a number of swellings resembling putty in appearance, but of fairly firm consistency, were observed. These areas were extensions through small perforations in the skull, from the dura which was extensively infiltrated. The brain was normal. A compact yellow mass about the size of a walnut rose up out of the sella turcica displacing the hypophysis but without eroding or distorting the clinoid processes. Another mass projected back from the right wing of the sphenoid which was extended to the ethmoids and both orbits." The histologic description of this tissue was, "striking yellow mass composed of spindle cells with large clear spaces filled with granular yellow substance. Scattered throughout the tumor were giant cells with rosette nucleus surrounded by a granular zone. The nodules from the dura showed the same changes. The entire pituitary was invaded by the yellow growth." Milne thought the condition was due to a tumor arising in the hypophysis. No report was made of the rest of the body. In a personal communication, Dr. Rowland said that tissue was examined by Dr. Lynch of New Zealand who reported "I am not able to say whether the condition is a primary tumor of the hypophysis or whether the invasion of the sella turcica is secondary.

8. Grosh, L. C., and Stifel, J. L.: Defects in the Membranous Bones, Diabetes Insipidus, and Exophthalmus, with Report of a Case, Arch. Int. Med. **31**:76 (Jan.) 1923.

9. Denzer, B. S.: Defects in the Membranous Bones, Diabetes Insipidus and Exophthalmos, Am. J. Dis. Child. **31**:480 (April) 1926.

10. Stowe, W. R.: Case of Diabetes Insipidus Associated with Defects in Skull, Tr. Australian M. Cong., September, 1927, p. 144.

The condition is best described as chordoma." Dr. Lynch considered xanthoma as a possible diagnosis.

In 1926, Kyrklund [11] published a case report and gave autopsy observations. The patient was a 12 year old girl who at 4 years of age developed thirst and polyuria. The cranium showed numerous defects. There was exophthalmos, but it excited no more interest or curiosity than had been shown in the study of other patients exhibiting the important syndrome, the outstanding clinical feature of which is exophthalmos. Autopsy revealed erosion of the skull and brownish-yellow material in the defective areas. The hypophysis did not show pathologic changes, but there were brownish-yellow growths in the brain stem behind the hypophysis, as occurred in other cases. The lungs showed extensive changes. Apparently no explanation was sought for the exophthalmos, but Kyrklund thought that the growths were of "sarcomatous" nature, and that the new growths in the region of the hypophysis accounted for the diabetes insipidus.

Schüller [12] manifested interest in the syndrome to which his name is sometimes attached, by reading before the first International Congress of Radiology in London in 1925, a short article on "Dysostosis Hypophysaria." In this article he reviewed his cases reported in 1915 and spoke of cases reported by Christian, Hochstetter and Alberti, but his interest did not carry him far into the literature or into the pathology of the condition. His summary was as follows:

> There exists a peculiar type of defects of the skull, characterized by the multi plicity, the great size and the sharp outlines of the defects localized as well on the cranium as on the base of the skull, combined with defects in the pelvis, exophthalmos, and with some of the symptoms generally diagnosed as due to pituitary underfunction, namely, dwarfism and diabetes insipidus. I propose for this symptom complex, the name Dysostosis Hypophysaria.

In 1928, Dr. R. S. Rowland of Detroit [13] reported in detail two cases of the syndrome under discussion, one of which came to autopsy. His first case was that of a white boy 5 years of age. Examination of the eyes showed "marked exophthalmos, greater in the right eye. The eye turned downward and inward. Pupils were slightly dilated, regular and equal, reacted to light and in accommodation. The muscle response was normal and nystagmus was not present. The fundi appeared nor-

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11. Kyrklund, R.: Beiträg zu einem seltenen Symptomenkomplex (Schadelerweichungen Exophthalmus, Dystrophia adiposogenitalis, Diabetes Insipidus), Ztschr. f. Kinderh. **41**:56, 1926.

12. Schüller, Artur: Dysostosis Hypophysaria, Brit. J. Radiol. **31**:156, 1926.

13. Rowland, R. S.: Xanthomatosis and the Reticulo-Endothelial System: Correlation of an Unidentified Group of Cases Described as Defects in Membanous Bones, Exophthalmos and Diabetes Insipidus (Christian's Syndrome), Arch. Int. Med. **42**:611 (Nov.) 1928.

mal; the discs were well defined and the vessels were normal in size and appearance." At autopsy, there was "marked exophthalmos of the type seen in cavernous sinus thrombosis. . . . Palpation of the skull revealed many cranial defects in which the bone appeared to be missing. . . . When the head was opened, the scalp was found normal in appearance. When the calvarium was exposed, there were numerous annular defects of the cranial bones which varied in size from 0.75 to 4 or 5 cm. in diameter. These defects had slightly wavy edges, but were fairly sharp. The bone at the margin was firmer than normal and the defect itself was filled with a gummy, semicaseous bright yellow tissue of rubbery consistency. . . . When the base of the skull was inspected, it was found irregularly covered over with these yellowish granulomatous swellings which had destroyed a large portion of the bone of the base, extending forward into both orbits and completely surrounding the region of the hypophysis and destroying the sella turcica. . . . The lungs were voluminous and felt fibrous to palpation. The left lung was somewhat adherent over the posterior lateral aspect of the costal pleura, the adhesions being of the same peculiar yellowish gummy nature that was noted in the granulomas of the skull." The pathologic report was by Dr. A. S. Warthin, who said that "the dural plaques consisted of masses of lipoid containing cells of reticulo-endothelial type resembling xanthoma. Throughout were numerous multinuclear giant cells. . . . These plaques did not suggest infectious granuloma but resembled xanthoma or masses of cells of the xanthoma type, and probably consisted of proliferated reticulo-endothelial cells with cholesterolosis. Fat stain showed cells to be loaded with lipoids." Dr. Warthin gave no report of examination of tissue from the orbit.

Rowland's second case was that of a white boy, aged 3 years, 11 months. In this case the ophthalmologic examination was made by Dr. Parker Heath:

> On April 24, 1926, examination showed vision in each eye, as determined by objects, normal for distance and near. Convergent power normal; no paralysis of extrinsic muscles. Pupils equal in diameter and react promptly to light and in accommodation; consensual reflexes are present. The left eye is slightly more prominent than the right, being proptosed approximately 2 mm. There is none of the lid lag sign found in exophthalmic goiter. Neither globe is congested. Ophthalmoscopic examination of the right eye: Pupils dilate evenly, media clear, disc normal in color, rings slightly blurred, vessels normal in caliber and course, foveal reflexes present. The left eye is essentially the same. The mechanism of the exophthalmos, judging from the x-ray plates, may be due to letting of the apex of the orbit as suggested by a previous observer.

Rowland thinks the cases represent a form of xanthoma in which many parts of the reticulo-endothelial system show storage of lipoid or

hyperplasia of lipoid cells. He discussed the group of cases described by different authors and expressed the belief that they represent the same condition. He said that "the researches on this subject indicate that all the varied manifestations of xanthoma can be brought back to the single pathologic principle that certain substances infiltrate the reticulo-endothelial system. The xanthoma cell is a cell of reticulo-endothelial origin infiltrated with lipoids. Xanthoma lesions are the hyperplastic reaction of the reticulo-endothelial system, resulting from the infiltration of lipoids in excess in the body fluids. A localization of this process produces the hyperplastic nodular lesions. . . . The study of this series of cases in which hyperplasia of the lipoid cells occurs both as a diffuse process in various organs and as localized nodular lesions clears up in a surprising way the mystery of the destruction of bone, exophthalmos, diabetes insipidus and dwarfism in the syndrome." Rowland also stated that "One is not dealing with true neoplasms, but with hyperplastic new formations, i. e., lipoid storage tumors. The formation of these nodules is a compensatory act on the part of the body in its attempt to rid the blood of an excess of lipoid which cannot be properly excreted."

In another article, published in June, 1929, Rowland [14] said, "In rapid development the infiltration is diffuse. In slow development the change is more localized assuming at times a tumor-like appearance. In the course of time, there is fibrosis, occasionally necrosis and cyst formation which mask the original picture."

In 1929, Hausman and Bromberg [1] reported in detail the early study of the patient who later was admitted repeatedly to the eye service of Bellevue Hospital, and who was operated on there. It is evident that the pathologic condition had been progressing over a period of three years, and the advance in the condition had revealed itself principally in the increased exophthalmos and in the pathologic ocular changes. The authors gave a careful review of the literature and made interesting comments on the sex, age, pathogenesis, pathology and symptomatology of the syndrome. They called attention to the difference of opinion as to whether diabetes insipidus is caused by a lesion of the posterior lobe of the pituitary body or one of the tuber cinereum, but they expressed the opinion that diverse views on this point can be reconciled, as the posterior lobe of the pituitary body, the stalk and the tuber cinereum in all probability constitute a physiologic as well as an embryologic and anatomic unit. They raise the question as to whether these para-infundibulum structures may not be responsible in some measure for the atrophic control of bone, and they hint that the pathologic condition

14. Rowland, R. S.: Christian's Syndrome and Lipoid Cell Hyperplasias of the Reticulo-Endothelial System, Ann. Int. Med. **2**:1277, 1929.

of the same structures may occasion the defects in the skull bones. They emphasize the high incidence of antecedent infection in the cases of Christian's syndrome which they studied. They say "particular significance is attached to the dysostosis in relation to the mechanism of diabetes insipidus."

REPORT OF CASE

History.—C. C., a 4 year old boy of Italian parentage, was admitted to the eye wards of Bellevue Hospital on Nov. 22, 1927. He had previously been admitted to Mount Sinai Hospital on July 19, 1927, and on Aug. 21, 1927. The dates of later admission to Bellevue on the eye service were Nov. 26, 1928, and April 22, 1930. During the period between November, 1927, and April, 1930, he was seen occasionally in the Bellevue outpatient clinic. At the time of his first appearance at Bellevue Hospital, the parents said that they had noticed the prominence of the right eye about two years before and that of the left eye only about a year later. The right eye had continued to be more prominent than the left, and the prominence had persisted in getting worse. The parents said that unusual thirst and frequent urination began about the same time that the right eye began to protrude. The father thought that the child was all right until he had measles, and that the present condition was due to the measles. Except for the prominence of the eye, the constant thirst and the frequent passing of urine day and night, the parents thought the boy normal.

Examination.—The child appeared rather apathetic, with an open mouth and listless expression. He carried his head slightly toward his right shoulder, and his right ear was noticeably lower than the left. He had a "froglike" expression, owing to the prominence of his eyes and the flattened appearance of his forehead. He was slightly stooped, and his abdomen was large. The genitalia were normal. He appeared to see and hear well, and he seemed normally bright for his age.

The motility of the eyes was good; the pupils were round, and the cornea, iris, lens and vitreous of each eye were normal. Examination was made under ether anesthesia. Tension (Schiötz) in the right eye was 18 mm. and in the left eye, 20 mm. The exophthalmometer readings were: right eye, 24 mm. and left eye, 23 mm. The fundi appeared normal, except for slight congestion of the retinal veins and slight edema at the disks. There was no measurable elevation. There was a moderate squamous blepharitis. The proptosed eyes could not be put back in position by pressure, and they did not pulsate.

Palpation of the head demonstrated areas of decreased resistance to pressure. The areas were not really soft, and seemed neither elevated nor depressed. No pulsation could be felt over the rarefied areas. No bruit could be heard with the stethoscope applied anywhere on the head.

The Wassermann tests of the blood and spinal fluid were negative. The urine was normal, except for low specific gravity (1.004). The blood was normal.

Roentgen examination showed striking defects in the bones of the skull, more on the right side than on the left. All of the bones showed material losses, but those about the right orbit and right temporal bone were most marked.

The little fellow went about the wards and corridors with a tincup in his hand, asking every one for a drink. He was always wet from free urination. He seemed content, apparently free from pain. He ate well and slept well, except that his thirst led him to waken occasionally and ask for a drink of water.

Second Admission (Nov. 26, 1928).—The general condition seemed about as before, but the exophthalmos had increased markedly and the epithelium of the

cornea of the right eye was broken. As sight was endangered, it was decided that the margins of the lids should be kept nearly approximated. Accordingly, the child was put under ether, and preparations for adhesions between the margin of the lids were made. The marginal epithelium was removed from corresponding areas on the upper and lower eyelids near the outer and inner margins of the corneas of both eyes. Sutures were introduced to hold the denuded areas in apposition.

While the patient was under anesthesia, the eyes were examined. The exophthalmometer readings were 35 mm. for the right eye and 33 mm. for the left. This represented a startling increase of proptosis in one year, as the readings in November, 1927, were 24 for the right eye and 23 for the left. The eyegrounds looked about as they did a year before, with a little edema at the papillae, but with too little elevation for measurement.

Roentgen examination showed larger defects of the bones of the skull, but none in the long bones.

The polydipsia seemed worse in that the child asked for water more often, especially at night. The specific gravity of the urine was 1.002. Under subcutaneous injections of solution of pituitary, the output of urine approached normal, and the abnormal thirst disappeared. It was not possible to get an accurate estimate of the amount of urine passed, because of the established uncertain habits of urination.

At this period the spinal fluid was found normal, and the Wassermann reaction was again negative.

The blood calcium and phosphorus were reported normal.

The operative procedure for the production of lasting adhesions to hold the eyes nearly closed was a success, and the patient peeped out through the slightly separated eyelids.

Third Admission (April 28, 1930).—At some time since the child was last in the hospital, one of the members of the attending staff cut the inner adhesion of the right eyelid, and partly liberated the eye (fig. 1). The right eye showed not only extreme proptosis, but marked depression as well, and there was little motility in any direction. The movements of the left eye seemed unimpaired. The pupils were semidilated and reacted only slightly to light. Lateral nystagmus had developed, so impairment of central vision was suspected.

Roentgenograms showed progression of the bony losses in the skull, and also a defect in the iliac fossa of the right innominate bone.

The Wassermann test of the blood was again negative.

The hemoglobin was 65 per cent. Erythrocytes numbered 3,345,000; leukocytes, 7,900; polymorphonuclears, 69 per cent; lymphocytes, 28 per cent; large monocytes, 3 per cent. The blood calcium was 12.1. The child weighed 27 pounds (12.2 Kg.).

The father had noticed that the boy was not seeing as well as formerly, so he urgently requested that one of the adhesions be cut to liberate the left eye, as had been done for the right eye. Ether was given, and the nasal adhesion on the left side was cut. Opportunity was again afforded for examination under anesthesia. The interior of each eye appeared almost identical. The cornea of the right eye showed slight opacity. Otherwise, the anterior structures were normal. The vitreous had dustlike and fine membranous opacities. The retina showed changes unobserved before. The veins were dilated and tortuous. The papillae were pale, but still slightly edematous. There were numerous punctate hemorrhages scattered through the background, but particularly in the macular regions, accounting for the apparent reduction of central vision and the nystagmus. Involvement of

Fig. 1.—Extreme exophthalmos of the right eye. Two adhesions have been introduced between the lid margins of each eye for protection of the globes and to prevent complete prolapse. The nasal adhesion on the right side has been cut.

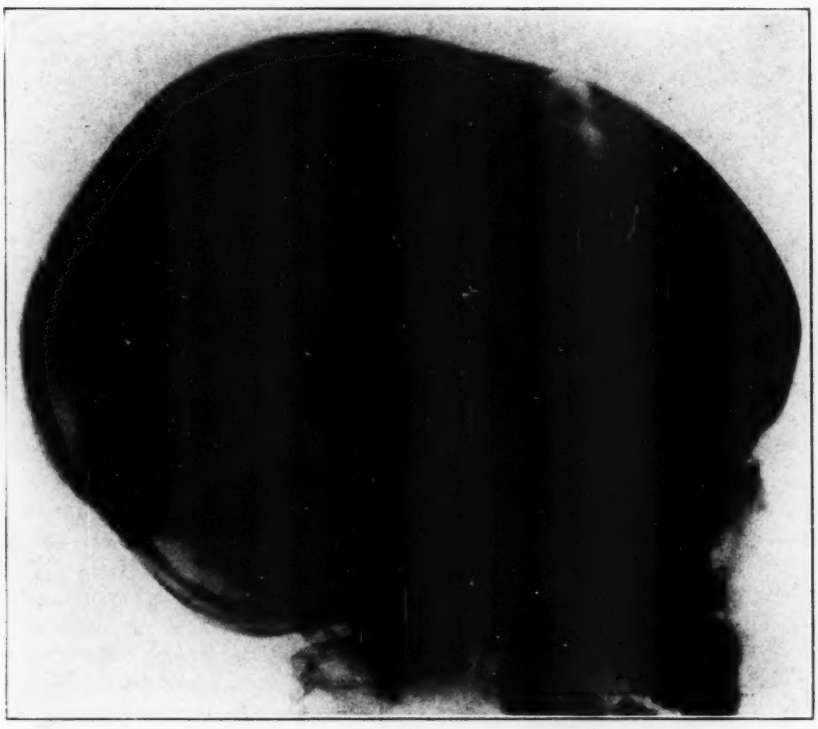

Fig. 2.—Roentgenogram of the skull showing large defects due to loss of bone, especially wholesale destruction of the bone in the orbital region.

the optic nerve was considered a factor, too. There were markings to indicate that the leakage had been going on for some time. The pupils were dilated and reacted only slightly to light. Palpation revealed a defect in the orbital margin above. Deep orbital palpation demonstrated a firm, nonpulsating, movable, nodular mass about 1 cm. in diameter resting on the floor of the orbit near the temporal wall. A smaller mass could be felt near the roof of the orbit. On the left side, a defect in the orbital margin and anterior part of the roof could be felt.

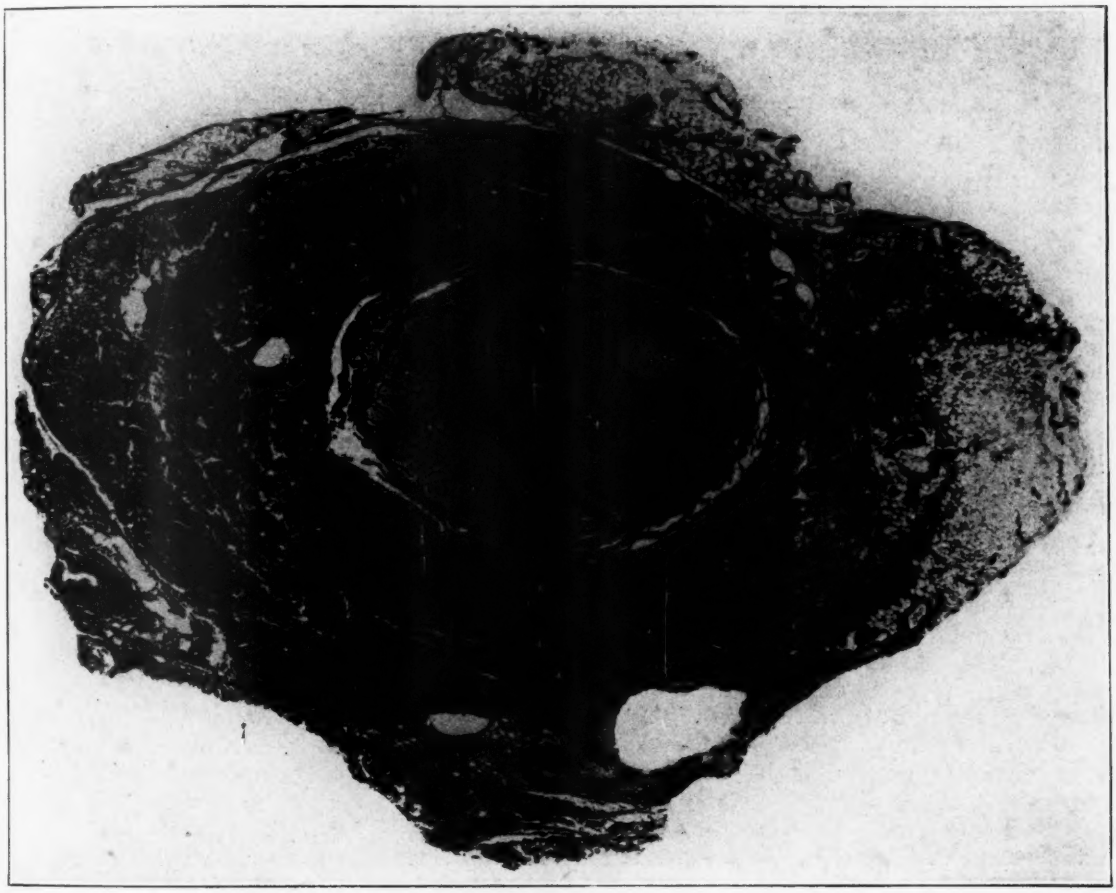

Fig. 3.—Section of the retrobulbar growth showing the optic nerve completely enclosed in a large nodule of newly formed tissue. The specimen was taken from the right orbit. (Preparation by Dr. A. E. Town.)

While the child was completely under ether, removal of some of the newly formed material from the right orbit seemed proper. A free incision was made along the orbital margin below, and the orbit was entered. Normal fat presented, but there was no difficulty in finding and removing the large nodule that had been felt on the floor of the orbit. Then it was found that the orbital cavity was nearly full of the extraneous nodular material, and that the optic nerve was completely enclosed by it. A piece was taken, including the nerve. The masses were distinctly yellow, and were rubbery to feel. There is no previous record of

excision of material from the orbit of a living victim of Christian's disease, but the material removed at operation answered to the description of that removed at autopsy in Rowland's case. It is worthy of note that the shape and size of the orbital cavity seemed normal, and that the periosteum seemed intact. While it was easy to determine that there was large loss of bone from the orbital walls, because of decreased resistance to pressure, it was plain that there was no bulging of periosteum into the cavity of the orbit, no pulsation and no evidence to support the accepted theory that loss of bone explained the exophthalmos. There was

Fig. 4.—Photograph of high power microscopic appearance of the tissue from a neoplastic-like nodule of the right orbit. It resembles postmortem tissue described by Rowland and Warthin (xanthomatosis). (Preparation by Dr. A. E. Town.)

definite evidence that the expanding newly formed masses had crowded the eyeball forward, just as a carcinoma, a cyst or an abscess would have done in the same orbit. Postoperative healing was uneventful.

Microscopic Report.—Dr. Douglas Symmers submitted the following report on the tissue: "Microscopic examination of the tissue removed from the eye in the case of C. C. shows the presence of an infiltrating tumor made up of large cells of variable shape with a sharply defined limiting membrane, a clear or finely

foamy cytoplasm, and a small, rather richly chromatic nucleus. In other words, the cells are morphologically identical with those of the so-called chordoma, but at the same time they resemble embryonal fat cells or xanthoma cells. Frozen sections of tissue removed from the tumor, however, and stained with sudan III, while showing large quantities of intercellular infiltrating fat, fail to reveal the slightest indication of fat or fatlike substances in any of the tumor cells themselves. For this reason, I think that the embryonal fat and xanthoma cells can be safely eliminated. The morphology of the tumor growth, together with its location at the base of the skull, combine, I think to establish the diagnosis of chordoma; that is to say, of a tumor arising from embryonal cartilage cells retained in the body as remnants of the notochord, which in the embryo consists of a rod of cartilage cells constituting the foundation of the axial skeleton. It extends throughout the entire length of the future vertebral column and reaches as far as the anterior end of the midbrain, where it ends in a hooklike extremity in the region of the future dorsum sellae of the sphenoid bone. The tumor springing from these embryonal cartilage rests is not at all common, but is perhaps most frequently seen as an infiltrative and destructive growth of the bones in the vicinity of the sella turcica and elsewhere in the skull."

In a personal letter, Dr. Russell S. Rowland said: "The section consists of a cross-section of optic nerve surrounded by granulomatous-like infiltration of the type characteristic of Schüller-Christian's disease. The infiltration consists of large lipoid-containing xanthoma cells, proliferating fibroblasts and lymphocytes. This case is identical histologically with that already reported by myself. It is of interest to mention that in the case described by Dr. Milne of New Zealand the pathologist's diagnosis was chordoma with xanthoma questioned. My own pathologist, Dr. Plinn F. Moorse, of Harper Hospital, states that the cells of a chordoma are quite different from these lipoid-containing cells, frequently resembling your cartilage cells."

COMMENT

Exophthalmos, large defects in the bones of the skull and diabetes insipidus make up a syndrome of interest to all clinicians. It is of especial interest to pediatricians because it occurs in children. It is of prime importance to ophthalmologists because the striking tell-tale sign is the exophthalmos, and because the optic nerve and retina may become involved, with consequent impairment of sight. The exophthalmos may be so great that impairment or loss of sight may result from exposure of the cornea.

Given a child with unilateral or bilateral exophthalmos without evident cause, the ophthalmologist may well think of the possibility of Christian's syndrome. Let him palpate the skull for possible defects in the bone, particularly around the orbit. Let him subject the patient to roentgen examination of the head and of the hip bones if he wishes, and the roentgenologist may demonstrate areas of loss of bone that cannot be discovered by palpation. Let him think of the possibility of excessive thirst, of excessive urine or of low specific gravity (diabetes insipidus). With the condition well in the ophthalmologist's mind, the diagnosis of the syndrome is easy.

Wassermann tests of the blood and spinal fluid have not been positive in any case. Tuberculin tests have been done in only a few cases, but there seems to have been no positive report.

Chemical examination of the blood reveals nothing characteristic. Examination of the cellular elements of the blood may show nothing abnormal. In this regard, there is quite a contrast with such a condition as chloroma.

At present one has a right to a mental picture of a neoplastic or neoplastic-like process involving structures in the floor of the third ventricle (tuber cinereum, infundibulum), possibly involving the hypophysis and other nearby cerebral structures, invading the orbit, and involving the bones of the skull and perhaps other bones. The infiltrating and destructive tissue need not be of a single pathologic class, but it is most likely to be the yellow, nodular, lipoid storage xanthoma that Rowland has held responsible for this vicious process which occurs in children.

With reasonable assurance, diabetes insipidus can be accounted for by disturbance of the cerebral tissue in the floor of the third ventricle, with or without invasion of the hypophysis. The defects of the bone probably are not the result of interference with function of these same cerebral structures, but are due to true invasion of the bones by the hyperplastic process. The pronounced exophthalmos is probably never due to mere loss of bone of the roof and other bony parts of the orbit, but to true invasion of the orbit by the hyperplastic process.

Invasion of the hypophysis may occur with or without erosion of the sella turcica. If the hypophysis is injured, asexuality, dwarfism and other evidences of interference of function may result.

OCULAR LESIONS IN SEPTICEMIA*

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A study of the eyes in thirty-two cases of septicemia obtained at autopsy has revealed a number of lesions not hitherto described in this condition. The cases of septicemia, as will be noted, were due to a variety of organisms. As a rule, only the posterior segment of the eyeballs was obtained for histologic examination. In this series no metastatic intra-ocular abscesses were found. Septic retinitis (Roth) was occasionally noted, but this subject has been dealt with so frequently that we have excluded our observations on this condition from our present report.

SEPTIC CHOROIDITIS

Marchesani,[1] in his interesting investigations on the pathogenesis of sympathetic ophthalmia, reported the following experiment. Pure cultures of avirulent organisms (*Staphylococcus albus* and *Bacillus xerosis*) were injected repeatedly, at intervals of two weeks, into the vitreous of one eye of a rabbit. In a number of instances it was possible to demonstrate an inflammatory reaction in the choroid of the uninoculated eye after the third or fourth injection. These lesions consisted in a loosely nodular accumulation of mononuclear cells of various types, rarely associated with local tissue necrosis. Cultures from these metastatic lesions failed to reveal any organisms, and Marchesani concluded, therefore, that the reaction was due to local allergy (he used the term "anaphylaxis"). Von Szily [2] repeated Marchesani's experiments and showed that similar lesions could be produced by repeated intravenous injection of cultures of the same organisms. He concluded that local allergy in one eye was not necessary for the appearance of the phenomenon in the fellow eye. The possible relation of these observations to sympathetic ophthalmia, therefore, remained doubtful.

It is of interest to discover that essentially similar lesions occur in the human choroid in septicemia. The lesions that we have found are somewhat less extensive than those described by Marchesani and von

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* From the Wilmer Ophthalmological Institute and the Department of Pathology of the Johns Hopkins Medical School.

1. Marchesani, O.: Arch. f. Augenh. **100-101**:606, 1929.
2. von Szily: Tr. Internat. Cong. Ophth., Amsterdam, 1929.

Szily, as is, perhaps, to be expected from the fact that in human septicemia the quantity of bacteria in the blood is not as great as in that in the experiments on animals reported, especially those of von Szily. In nine cases, enumerated in the accompanying table, foci

Fig. 1.—Septic choroiditis in a case of hemolytic streptococcus septicemia.

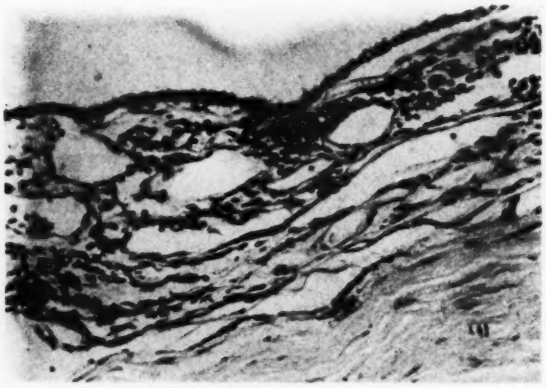

Fig. 2.—Septic choroiditis in a case of gonococcal endocarditis.

of infiltration were seen in the choroid. When present, these infiltrations were always bilateral. They consisted in loose accumulations of mononuclear wandering cells in the stroma of the choroid. The predominant cell type was the small lymphocyte, but large lymphocytes, large mononuclear cells resembling the transitional cells of the blood stream, were also seen in numbers. Plasma cells and epithelioid cells

occurred more rarely. The lesions were never associated with local tissue necrosis or hemorrhage, nor was there ever any involvement of the overlying retina. In view of the striking uniformity of the histologic picture in these cases, only two instances are reproduced in the accompanying illustrations (figs. 1 and 2).

The lesion that we have described is of no great clinical or pathologic importance. It occurs late in septicemia and does not, we believe, produce any striking ophthalmoscopic changes—at least we have observed no such changes. It is analogous to similar infiltrations found in the looser and more cellular mesodermal structures throughout the body in septicemia, such as the spleen and lymph glands, the periportal spaces in the liver and the perivascular spaces in the hilus of the kidneys. Its significance is derived from the light which it sheds on the experiments of Marchesani and von Szily, supporting the interpretation of the

Anatomic Condition Causing Septicemia in Nine Cases

Case	General Anatomic Diagnosis	Organism Causing Septicemia	Figure
1	Pelvic abscess, rupture into iliac vein.....	Hemolytic streptococcus	1
2	Subacute bacterial endocarditis.....	Peculiar hemolytic streptococcus	
3	Hodgkin's disease, peritonitis.....	Hemolytic streptococcus	
4	Sinusitis, meningitis, infection of superior longitudinal sinus	Staphylococcus aureus	
5	Pneumonia, acute endocarditis, meningitis	Pneumococcus type II	
6	Epithelioma of bladder, pyelonephritis....	Bacillus coli	2
7	Acute endocarditis.....	Gonococcus	
8	Acute endocarditis.....	Gonococcus	
9	Erysipeloid infection of skin.....	Streptococcus pyogenes	

latter, as against that of the former, in indicating that the so-called "allergic choroiditis" of Marchesani has nothing to do with the etiology of sympathetic ophthalmia.

We are unable to state whether the lesion is due to an actual bacterial implantation in the choroid or to a local proliferation or deposit of wandering cells in response to the general demands of the body in septicemia. The negative results of culture in Marchesani's experiments do not disprove the former theory, for the avirulent organisms which he used would promptly die in the tissues. Nor would positive cultures in our cases have proved the contrary, for the blood stream in our cases was heavily infected with virulent organisms, and no proof would have been available to determine whether organisms obtained on culture had been derived from the blood stream or from the choroidal lesion. We did not, therefore, sacrifice any of our histologic material for bacteriologic examination. The varied type of the infiltrating cells that we found and the absence of any germinal forms may be taken as evidence against the theory that the cellular accumulations are due to local proliferation.

Bacterial stains in our cases revealed bacteria within the vessels of the choroid and also in the stroma of the choroid in close association with the lesions that we have described, and also in places in the stroma where no cellular proliferation was found. It is possible, therefore, that the bacteria in the stroma may have grown out of the infected blood channels post mortem.

CHOROIDITIS AND NEURORETINITIS IN ENDOCARDITIS LENTA

The picture of endocarditis lenta is now generally recognized as a clinical entity. The organisms that are held responsible for this condition often belong to the *Streptococcus viridans* group. According to Libman, the chief features of this disease are marked progressive anemia, brown pigmentation of the face, renal disease, splenic enlargement and endocarditic symptoms, such as fever, embolism, arthritis and petechiae. On the basis of these general symptoms, an attempt has been made to correlate this disease with a chronic septic uveitis, which Gilbert [3] called "ophthalmia lenta." He collected nine such cases from the literature. Von Herrenschwand [4] upheld the relationship between these two conditions. According to him, ophthalmia lenta is a recurrent bilateral iridocyclitis with hypopyon. The disease occurs in young adults at the end of some general infection, particularly after inflammation of the joints. Blüthe,[5] Gilbert and Weve [6] investigated five cases histologically, finding the most severe changes in the anterior vitreous, where, as a result of the repeated inflammation, scar tissue masses were found. Perivascular and nodular lymphocytic infiltrations were also found in the choroid. Weve denied that this condition has any relationship to endocarditis lenta, and attributed it to an allergic reaction to the staphylococcus.

More characteristic of endocarditis lenta is the so-called "septic retinitis" first described by Roth, in which petechial hemorrhages with yellowish or white centers are the characteristic ophthalmoscopic features. The following case presents an extraordinary and, we believe, hitherto undescribed ocular complication of endocarditis lenta.

CASE 1.—A colored girl, aged 15, was admitted to the medical service of the Johns Hopkins Hospital on March 25, 1929, complaining of a heavy cold and pain in the stomach. The family history was unimportant. The patient had suffered from a variety of acute infections in infancy and early childhood. There was no history of rheumatic fever. Three months prior to admission, after repeated upper respiratory infections, she became dyspneic and complained of precordial pain. Pain and tenderness soon developed and migrated from one joint to another.

3. Gilbert: Arch. f. Augenh. **96**:119, 1925.
4. von Herrenschwand, F.: Klin. Monatsbl. f. Augenh. **83**:422, 1929.
5. Blüthe: Inaug. Diss., Heidelberg, 1908.
6. Weve: Arch. f. Augenh. **93**:14, 1923.

Examination.—The temperature was 101.8 F., the pulse rate, 130, and the respiratory rate, 24. The blood pressure was 120 systolic and 70 diastolic. Examination showed conjunctival petechiae, subcutaneous edema, cyanosis and pharyngeal redness. Râles were present at the base of the right lung. The heart was enlarged, produced a systolic murmur and showed signs suggestive of adhesive mediastinitis. There was tenderness over the liver and spleen, with increased splenic dulness. The knee joints were tender and warm and contained fluid. Examination of the blood showed: red cells, 3,720,000; hemoglobin, 55 per cent; white cells, 13,300, with 81 per cent polymorphonuclear cells. Urinalysis showed: albumin +, red and white blood cells + and urobilin ++. The Wassermann reaction was negative. After repeated cultures, *Streptococcus viridans* was isolated from the blood. The phthalein excretion was 30 per cent in two hours, but there was no nitrogen retention.

Course.—While the patient was in the hospital signs of aortic insufficiency and later of cerebral embolism developed. Lumbar puncture was performed, and the spinal fluid was found to be under increased pressure and contained 550 red blood cells and 64 white blood cells. No organisms could be grown on a culture from

Fig. 3 (case 1).—Edema and perivascular infiltration of the optic papilla.

the spinal fluid. The patient's condition grew steadily worse, with a gradual rise of temperature and an increased pulse rate. Moderate generalized edema developed, and the anemia steadily increased. Her neck became very stiff, and the tendon reflexes in the arms became hyperactive. During the last ten days, multiple hemorrhages developed. The nonprotein nitrogen two days before death rose to 64. The patient died on May 20.

Medical Impression.—The diagnosis was rheumatic heart disease with enlarged heart, mitral insufficiency and stenosis, aortic insufficiency, adhesive mediastinitis, myocardial failure, endocarditis lenta, acute toxic nephritis and cerebral infarct.

Ophthalmoscopic Examination.—The eyes were examined repeatedly during the patient's stay in the hospital. They presented a picture which changed very little during the course of the illness.

In the right eye there was marked generalized neuroretinal edema. The disk was hyperemic and projected forward 5 diopters, with its margins almost totally obliterated and its cup practically entirely abolished. The veins were moderately engorged, and the lower temporal vein appeared to be thrombosed, with numerous hemorrhages along its course. The larger arteries were somewhat attenuated, but patent. All of the vessels were somewhat obscured by the generalized edema. Scattered along the smaller vessels, yellowish-gray stripes were found, which

sometimes coalesced over the surface of the vessel, obscuring the blood column. These were seen along both the arteries and the veins.

The left eye presented an essentially identical picture.

General Anatomic Observations.—At autopsy the heart was found enlarged and showed a florid vegetation on the mitral valve. There was a fresh, fibrinous pericarditis. There were infarcts in the spleen, kidneys and brain and a thrombosis with mycotic aneurysm in the hepatic artery. There were several minute abscesses in the heart muscle and an embolic nephritis. *Streptococcus viridans* was grown on a culture from the heart's blood.

Ocular Pathology.—Only the posterior segments of the globes were obtained for microscopic study. The observations in both eyes were identical. The optic papilla and surrounding retina were edematous. There was some serum and mononuclear cell infiltration of the vitreous, concentrated especially over the disk, which showed marked perivascular round cell infiltration. Scattered retinal and preretinal hemorrhages were found. The most remarkable features were seen in connection with the smaller retinal vessels, which showed in many places oblitera-

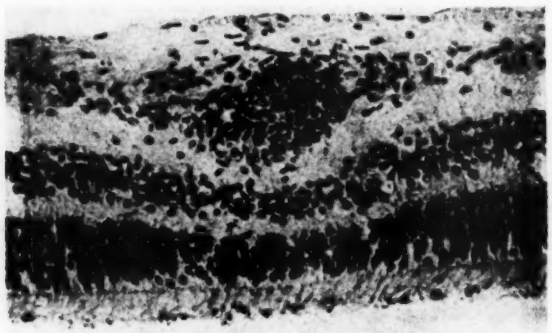

Fig. 4 (case 1).—Purulent focus in the retina.

tion of their lumina, with intense wandering cell infiltration of their walls. In what appeared to be the fresher of these lesions, the infiltration into and about these vessels was mainly polymorphonuclear, suggesting the appearance of minute embolic abscesses. In the older lesions the infiltration was mainly mononuclear, consisting equally of small lymphocytes and larger mononuclear cells resembling epithelioid cells. In some places these infiltrates had burst through the surface of the retina and formed small domelike masses in the vitreous. Strands of fibrin were found within the occluded vessels. Some of the wandering cells contained gram-positive cocci. These lesions, followed in serial section, were definitely nodular and of small extent, though in the adjacent portion of the vessels perivascular infiltrates of lesser grade were seen. The choroid and optic nerve behind the papilla showed no lesions.

Comment.—So far as we are aware, no case similar to the one just reported has ever been noted either in subacute bacterial endocarditis or in acute rheumatic fever. At first sight the lesions present a superficial resemblance to the Aschoff bodies found in various tissues in rheumatic fever. This resemblance merits discussion because of the recent

recrudescence of the theory that acute rheumatic fever is caused by *Streptococcus viridans*. Against the interpretation of these lesions as Aschoff bodies are the following facts: The earliest lesions in our case are apparently those made up of polymorphonuclear infiltrates about occluded vessels. Such polymorphonuclear infiltrates are not seen in rheumatic fever. The more developed lesions contain mainly mononuclear cells, but plasma cells are not present, while, on the contrary,

Fig. 5 (case 1)—Organizing inflammatory nodule in the retina.

Fig. 6 (case 1).—Inflammatory nodule extending into the vitreous.

they are the main elements of the Aschoff bodies. Finally, no evidence of fresh acute rheumatic fever was found in any other organs in this case. It seems clear, therefore, that we must attribute the ocular lesion to the bacterial endocarditis and not to an underlying acute rheumatic fever. In view of the histologic observations, it seems most reasonable to interpret these lesions as embolic.

The occurrence of isolated embolic occlusions of retinal vessels in bacterial endocarditis is well known clinically, but there is usually little evidence of inflammatory reaction about the embolus.

CASE 2.—A colored woman, aged 40, was admitted to the medical service of the Johns Hopkins Hospital on Oct. 4, 1923, complaining of headaches, dizziness and general weakness. The patient's mother and sister had died of heart and kidney disease. The past history was unimportant except for two miscarriages. The patient had suffered with nocturia for some time, severe headaches with vomiting and dizziness, and increasing dimness of vision for four months prior to admission, associated with dyspnea on exertion.

Examination.—There was marked dilatation of the heart with signs of aortic insufficiency and roughened aorta. The blood pressure was 280 systolic and 150 diastolic. The temperature was 97.8 F. There was a secondary anemia. The urine showed albumin, ++++, with a phthalein excretion of 30 per cent in two hours, and a nonprotein nitrogen of 40. The Wassermann reaction was 1 plus. *Streptococcus viridans* was obtained repeatedly on a culture from the blood. The patient died suddenly ten days after admission with signs of intracranial hemorrhage.

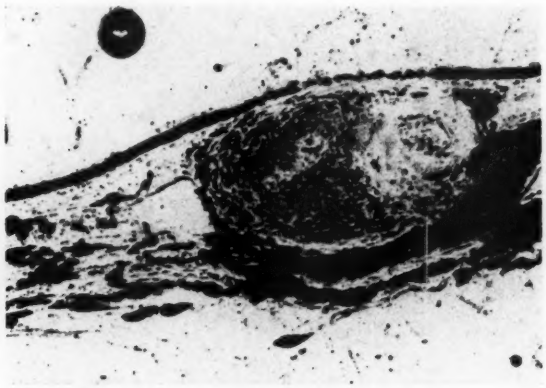

Fig. 7 (case 2).—Periarterial nodule in the choroid.

Medical Impression.—The diagnosis was: hypertension; arteriosclerosis; chronic nephritis; syphilitic aortitis with aortic insufficiency; *Streptococcus viridans* septicemia.

Ophthalmoscopic Examination.—The picture was typical of albuminuric retinitis, with extreme sclerotic changes in the retinal arteries. In the macular region of the right eye, three rounded yellowish elevations were seen, which were apparently beneath the retina, as is well pictured in figure 7.

General Anatomic Observations.—At autopsy there were found: syphilitic aortitis, cardiac hypertrophy, chronic nephritis, moderate sclerosis of the cerebral vessels and a hemorrhage into the cerebellum. The smaller arteries in all of the organs showed extreme fibrous thickening of their coats, with some atheromatous changes in the intima. There was moderate hyaline degeneration of the arterioles, but no perivascular inflammatory lesions except those found in the eyes.

Ocular Pathology.—Only the posterior segments of the globes were obtained for microscopic study. The left eye showed extensive retinal arteriolar sclerosis, with typical albuminuric retinitis. The right eye, which was embedded in gelatin and stained for fat, showed the same changes, and, in addition, the three oval nodules of inflammatory tissue were found in the choroid. The smallest of these

was approximately 0.1 mm. in diameter, the largest being about four times this size. All three lesions appeared definitely related to the sclerotic and partially thrombosed arterioles. The lesions consisted of an organized inflammatory mass made up of fibroblasts and large mononuclear wandering cells. No giant cells were seen, and the lesions did not resemble tuberculous or syphilitic reactions. They were sharply circumscribed, with only slight mononuclear infiltration of the surrounding choroid.

Comment.—In this case *Streptococcus* viridans septicemia was a terminal event superimposed on a complicated background of disease. The inflammatory nodules in the choroid might be attributed to the syphilis, to the arteriolar sclerosis and chronic nephritis, to the terminal septicemia or to some obscure unrecognized infection. Against the syphilitic origin of these nodules is their histologic picture, which in no way resembles the familiar picture of syphilitic choroiditis. Furthermore, the syphilitic infection in this case was an old one, as evidenced by the lesions in the aorta, and was therefore not in the stage when disseminated choroiditis is to be expected.

Hanssen and Knack [7] reported a large group of cases of nephritis with and without albuminuric retinitis, in which choroidal inflammatory nodules were found, practically identical in appearance to those in case 2. Their investigations were made during the World War, at one of the large German base hospitals. On the basis of their observations, they have formulated the theory that albuminuric retinitis is essentially an inflammatory disease not directly related to arteriolar sclerosis. The conclusions of these writers have been questioned by many investigators who have failed to find choroidal inflammatory lesions in large numbers of cases of albuminuric retinitis. We have likewise examined histologically thirty-five cases of albuminuric retinitis exclusive of the one just reported (case 2) and the one to follow (case 3), and in none of these have we found choroidal inflammatory lesions, while all exhibit retinal arteriolar sclerosis. We are forced to conclude, therefore, that some exceptional complication occurred both in the cases of Hanssen and Knack and in the one just reported (case 2), and we are inclined to attribute the lesion in our case to *Streptococcus viridans* septicemia. Many of Hanssen and Knack's cases were instances of trench nephritis, while others showed relapsing fever.

Goldstein and Wexler [8] described an almost identical lesion in a case of periarteritis nodosa with subacute endocarditis and multiple emboli in various organs. It is not clear from their description whether the ocular lesion is to be attributed to the underlying endocarditis or to the periarteritis nodosa. In view of the frequent association of

7. Hanssen and Knack: Klin. Monatsbl. f. Augenh. **59**:263, 1917.

8. Goldstein, H., and Wexler, D.: Ocular Pathology of Periarteritis Nodosa, Arch. Ophth. **2**:288 (Sept.) 1929.

albuminuric retinitis and chronic nephritis with periarteritis nodosa, we examined the tissues in case 2 with great care, searching for evidence of this disease. Though extreme forms of arteriolar sclerosis and arteriosclerosis were discovered, no periarterial nodules were found that would justify this diagnosis. The ocular lesion, however, resembles that described by Goldstein and Wexler so closely that we are inclined to believe that the two conditions are related. The final judgment as to whether the lesions are due to the septicemia or to an aberrant form of periarteritis nodosa must be withheld until the etiology of the latter disease has been established.

OCULAR LESIONS IN PERIARTERITIS NODOSA

Periarteritis nodosa is a disease of the blood vessels characterized by nodular thickening of the arteries. Because of the diversity of its symptomatology, resulting from the involvement of the various organs, the disease frequently is unrecognized during life. The typical histologic changes in the arterial walls appear in the early stages as areas of marked hyaline degeneration of the media associated with lymphocytic infiltration. Somewhat later, leukocytes, plasma cells and round cells involve the adventitia, and later there is a proliferation of the endothelium causing a contraction of the lumen of the vessels. The elastic tissue is frequently entirely destroyed. In the resulting thrombosis there is a considerable amount of scar tissue formation, causing the vessels to become cordlike at these points.

Many investigators have held that periarteritis nodosa is infectious in origin. Bacteriologic investigation of the blood and diseased tissue has brought forth many organisms, chiefly staphylococci and streptococci. Gruber, who has done a great deal of work on this disease, concluded that it is simply the reaction of the arterial system during the course of diverse infectious diseases.

CASE 3.—*History.*—A white man, aged 55, was admitted to the medical service of the Johns Hopkins Hospital on Dec. 1, 1928, complaining of nervousness and fever. The family and past histories were essentially unimportant. The present illness began with an attack of "mumps" eighteen months before admission, which was followed one month later by orchitis and prostatitis. Shortly afterward he began to lose weight and to have fever in the afternoon, which persisted with occasional nocturnal rigors.

Examination.—Examination gave essentially negative results, except for fever and loss of weight. The blood count, blood chemistry, urine and spinal fluid were normal. The Wassermann reaction was negative. Repeated blood cultures showed no growth. After two months in the hospital, the blood pressure, which had been normal on admission, began to rise, and the urine showed albumin, casts and white blood cells. About the same time a small tender subdermal nodule appeared on the arm, which, on excision, was found to contain chronic inflammatory tissue.

After four months in the hospital, cardiac decompensation developed, and the patient died of a terminal pneumonia. The diagnosis of periarteritis nodosa was not established clinically.

Ophthalmoscopic Examination.—Shortly after admission no lesions were visible in the fundi. Three months later the patient complained of blurring of vision, and ophthalmoscopic examination showed edema of the retina and optic disk, with marked retinal arteriosclerosis, arteriovenous compression, numerous hemorrhages and cotton-wool spots in both eyes.

Fig. 8 (case 3).—Periarterial nodule in skeletal muscle.

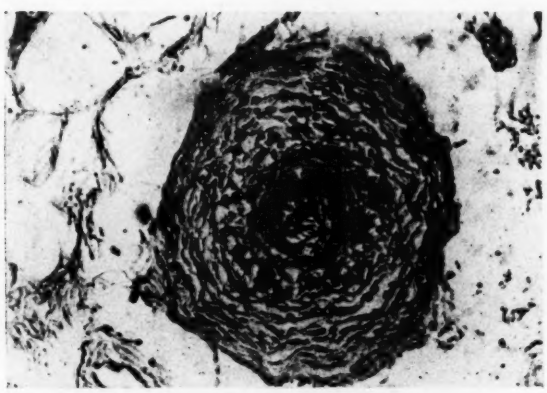

Fig. 9 (case 3).—Periarterial nodule in short ciliary artery just outside of the sclera.

General Anatomic Observations.—At autopsy there were found slight cardiac hypertrophy and advanced chronic nephritis, with small discrete areas of apparently normal kidney tissue persisting. The aorta showed some atheromatous plaques. The smaller arteries in all the tissues were studded with minute yellowish-gray nodules, from 1.5 to 2 mm. in diameter. On section these nodules showed hyaline degeneration of the media, with instense proliferation of fibrous tissue in the adventitia and under the intima, resulting in many cases in complete occlusion of the vessels.

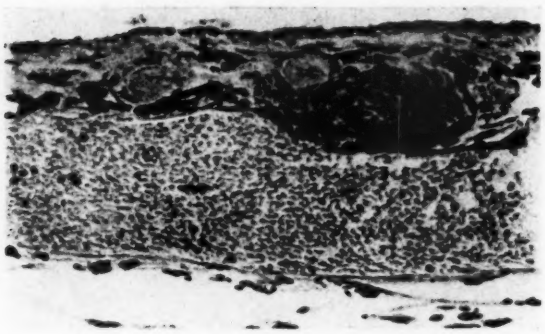

Fig. 10 (case 3).—Periarterial nodule in the choroid.

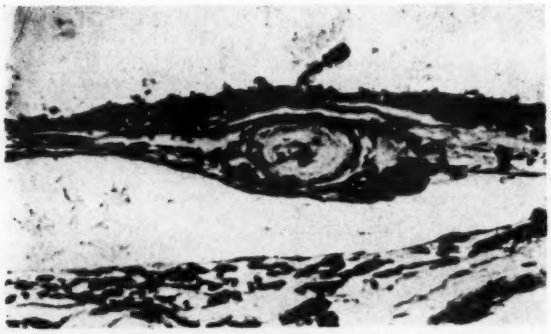

Fig. 11 (case 3).—Arteriolar sclerosis in the choroid.

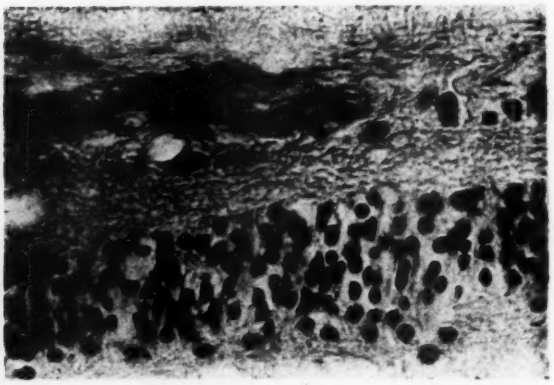

Fig. 12 (case 3).—Arteriolar sclerosis in the retina.

Ocular Pathology.—There was edema of the retina and optic disk. The retina showed an extreme arteriolar sclerosis with hemorrhages and serous exudates, patches of cytoid bodies and areas of infiltration with fat droplet cells, resembling in these respects the typical picture of albuminuric retinitis. No inflammatory lesions were seen in the retina. The choroid likewise showed extreme arteriolar sclerosis, but in addition a few minute vessels showed fibrous thickening of their coats with intense mononuclear infiltration, suggesting the appearance of the typical periarterial lesions seen in the larger vessels in other organs. Along the course of the short ciliary arteries, just outside the sclera, several typical nodular thickenings with fibrosis of the adventitia and infiltration with mononuclear cells were found. The optic nerve was normal. The central retinal vessels showed sclerosis but no inflammatory reaction.

Comment.—The clinical history and anatomic observations in this case leave no room for doubt as to the diagnosis of periarteritis nodosa. This is the first case of this disease, complicated by albuminuric retinitis, in which the eyes have been subjected to histologic examination.

Böck [9] and von Herrenschwand [4] each reported a case of periarteritis nodosa with typical nodular lesions in the short ciliary and other orbital arteries, but Goldstein and Wexler's is the only case previously reported in which marked intra-ocular inflammatory lesions were discovered. The atypical nature of the lesions described by Goldstein and Wexler and their similarity to the condition described by Hanssen and Knack, and by ourselves in case 2, have already been noted. Herrenschwand and others have drawn attention to the frequent occurrence of the ophthalmoscopic picture of albuminuric retinitis in periarteritis nodosa, and Herrenschwand discussed the interesting question as to whether or not this ophthalmoscopic picture in these cases is due to inflammatory lesions of the retinal arteries.

We are fortunate to be able to answer Herrenschwand's question in regard to the pathogenesis of the albuminuric retinitis in this condition. We found no evidence of inflammatory lesions in the retinal arteries; on the contrary, the retinal vessels show an extreme degree of arteriolar sclerosis, and do not differ in any way from those found in ordinary uncomplicated cases of albuminuric retinitis.

In the absence of knowledge of the specific etiology of periarteritis nodosa, one is perhaps justified in speculation as to the relation of this disease to other vascular lesions, and to suggest the possibility that the condition described by Hanssen and Knack and exemplified in case 2 may be a related form. The case reported by Goldstein and Wexler forms a bridge between these two conditions in that the choroidal lesion resembles that in the case recorded by Hanssen and Knack, while the visceral lesions are typical of periarteritis nodosa.

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9. Böck, J.: Ztschr. f. Augenh. **69**:225, 1929.

SUMMARY

1. Of thirty-two cases of septicemia due to a variety of organisms, nine cases showed an inflammatory lesion in the choroid, which we call septic choroiditis. This lesion resembles that produced experimentally in rabbits by Marchesani and von Szily, so-called "allergic" or "anaphylactic choroiditis." The finding of this condition in human cases without any clinical or histologic evidence of sympathetic ophthalmia strongly supports the view that these lesions are in no way related to sympathetic ophthalmia.

2. A case of subacute bacterial endocarditis complicated by embolic neuroretinitis is reported.

3. A case of chronic nephritis, arteriolar sclerosis, syphilitic aortitis, albuminuric retinitis with terminal *Streptococcus viridans* septicemia is reported. Periarterial inflammatory nodules were found in the choroid similar to those described by Goldstein and Wexler in a case of periarteritis nodosa and by Hanssen and Knack in cases of albuminuric retinitis. The possible relation of these two conditions is discussed.

4. A case of generalized periarteritis nodosa with ocular lesions is reported.

THE PATHOLOGY OF CHRONIC SIMPLE GLAUCOMA *

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It is not my purpose in this paper to offer a complete exposition of the pathology of glaucoma, but merely to present some practical examples, based on pathologic observations in glaucomatous eyes, together with deductions drawn from the comparative study of its special pathology with general pathology, of which it is only a part.

If the numerous opinions of different writers on glaucoma are reviewed, it will be found that each endeavors to demonstrate that glaucoma is produced by some special factor which he favors, and that all manifestations associated with the disease are made subservient to this favored cause. Thus, some writers invoke purely anatomic causes, as for instance, those who explain the pathology of glaucoma as associated with unusually small eyes, with special conformation of the anterior chamber and iridocorneal angle or with the changes that occur in the size of the lens as the result of age, etc.

Others claim that glaucoma results from different anatomopathologic lesions, such as local and general arteriosclerosis, with vascular lesions (atheroma, endophlebitis and periphlebitis), peripheral synechiae, closure of Schlemm's canal, sclerosis of the ligamentum pectinatum, primary degeneration of the optic nerve, etc.

According to still other writers, glaucoma results from purely functional disturbances observed in connection with vasodilatation and a subsequent increase in the quantity of aqueous humor due to different causes, with dysfunction of the endocrine glands, with physicochemical phenomena due to osmotic interchanges between the blood plasma and the intra-ocular fluids, with changes of metabolism and with engorgement of the vitreous and lens which has, as a mechanical effect, compression of the iris against Schlemm's canal and obstruction of the intraocular circulation, producing hypertonia, etc. Many other theories could be presented, but they may be found in the writings of research workers

* Submitted for publication, May 29, 1930.

* From the Chicago Eye, Ear, Nose and Throat Hospital.

* Read before the State Medical Association of Texas, Mineral Wells, May 7, 1930.

and in the recent publications of well known pathologists, as well as in textbooks devoted to the special pathology of the eye.

From the biologic standpoint, the eye is one of the most highly differentiated organic structures in the body. Considering, also, the complexity of the phenomena associated with the execution of the most simple function in any part of the body, it seems puerile to conclude that glaucoma is due to one factor only. From the eclectic point of view, no one case or group of cases can explain the generality. One cannot fall into the logical error of reasoning from the particular to the general. In searching for the pathologic basis of glaucoma it would seem more logical to consider the many factors that are associated and that work together to maintain normal function. When one or more of these factors is altered, equilibrium is still maintained by the compensatory mechanism that substitutes for the deficient functioning of some or the excess functioning of others. It is only when the changes in the factors are so extreme that the limit of possible compensation is passed that the pathologic state is established. Thus it is not by the exclusive influence of any one factor that the glaucomatous state is brought about, but through the association of many factors which ordinarily work in unison to maintain the perfect functioning of the whole organism.

In chronic simple glaucoma, a disease occurring in old persons, the whole organism presents generalized angiosclerosis; this sclerosis is not limited to the vessels alone, but is shared by several other structures, the regulatory mechanism being much diminished at this period of life as compared with juvenile elasticity of tissue. These subjects present a generalized degeneration corresponding exclusively to their senility.

This generalized sclerosis is also manifested in the intra-ocular vessels, arteries, capillaries and veins, which present atheromatous degeneration. In eyes with this condition nutrition is diminished, and there are all the other senile manifestations in the form of a generalized sclerosis in the stroma of all the structures, especially in the other vascular tissues. This sclerosis is more prominent in those places in which connective tissue is normally found, as in the optic nerve, in the entrance of the central vessels, in the exit of the venae vorticosae, in the spaces of Fontana and in Schlemm's canal. Other manifestations are hyaline degeneration, lipoid infiltration, and pigment emigration of the ectodermic layer of the ciliary body and iris, especially the latter, owing especially to faulty nutrition when there are no inflammatory processes present.

These changes in the vessels raise the blood pressure, especially in the capillaries and, at the same time, produce the angiospasm com-

monly observed in persons with arteriosclerosis, as the rigid walls do not give the normal response to vasomotor influence. In this way, a vicious circle, nervous-endocrine-humoral, is created with a progressive decrease in the metabolic exchanges or nutrition; indeed, the exact relation between these three factors is not known. One does not know whether the chemical composition of the organic humors regulates the nervous system and endocrine glands, whether the endocrine glands regulate the other two or whether the nervous system regulates the whole complex mechanism.

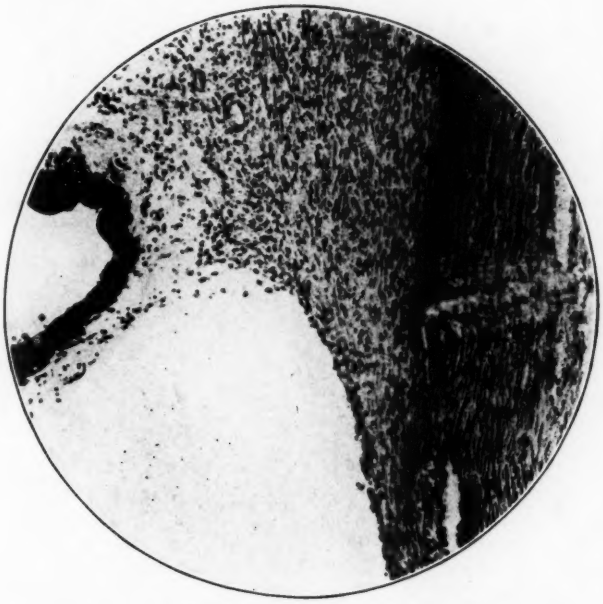

Fig. 1.—Angle of the anterior chamber in a case of myopia.

At the same time, the vascular walls with microscopic lesions undergo changes in their capacity as semipermeable membranes, and the osmotic equilibrium between the blood pressure and intra-ocular fluids is altered, more colloids being passed into the eye and the salts being diminished. If the osmotic equilibrium is altered, the nutrition of the vitreous body and lens is changed by the sclerosis of the hyaline membrane in the former and by massive sclerosis in the latter, the volume of both being increased and a state of turgescence being produced which pushes the iris forward, narrowing the anterior chamber.

I shall now analyze some anatomic dispositions that indicate why some eyes are predisposed to glaucoma and why others are not. Figure 1 shows the iridocorneal angle of a myopic eye in which the insertion of the iris is high on the ciliary body and distant from the spaces of Fon-

Fig. 2.—Angle of the anterior chamber in a case of hypermetropia.

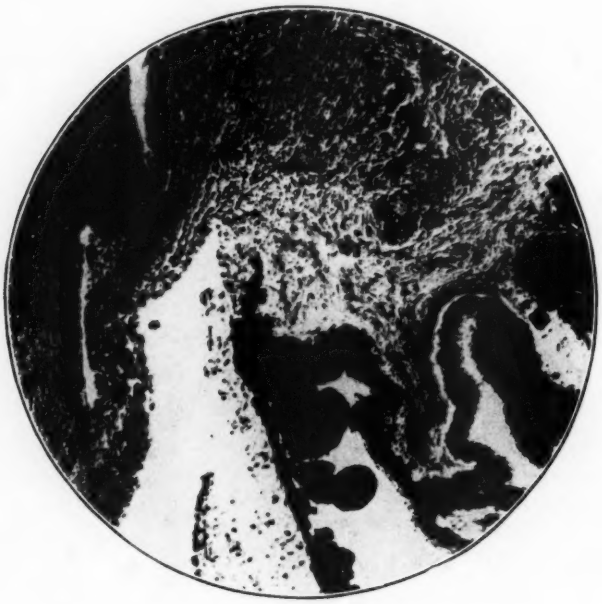

Fig. 3.—Another eye showing hypermetropia.

tana. There is accommodation for near work; the ciliary muscle does not function, but tends to atrophy. Moreover, in myopia the ciliary body has a peculiar position, being situated in front of the insertion of the iris and far from the cornea, which results in a very deep anterior chamber. The circular fibers of the ciliary muscle have disappeared and the muscle consists of longitudinal fibers alone, most of which also have undergone some degree of atrophic change.

Owing to the weakened ciliary muscle and the deep anterior chamber, the excursion of the iris, which depends on the expansion of the

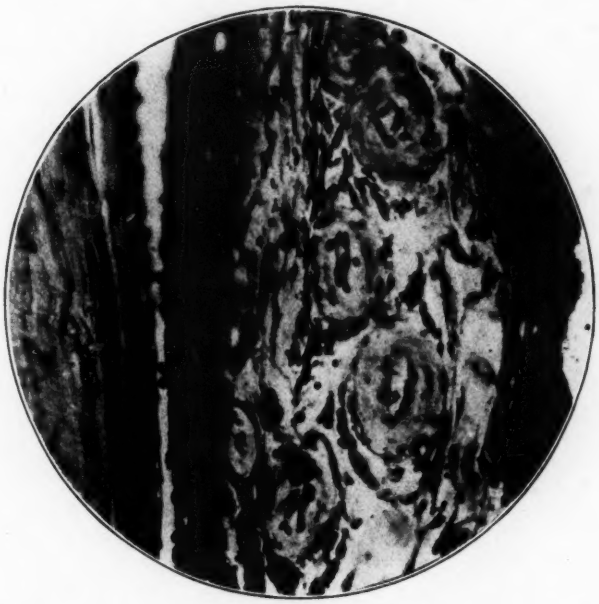

Fig. 4.—Sclerosis of the vessels of the iris. Emigration of pigment toward the anterior surface of the iris.

vitreous and lens, necessarily has to be much more extensive than normal in order to occlude the iridocorneal angle. An eye with this condition is therefore not predisposed to glaucoma.

On the other hand, the slides of hypermetropic eyes (figs. 2 and 3) show that the insertion of the root of the iris is much nearer to the spaces of Fontana with a consequent shallow anterior chamber. The circular fibers of the ciliary muscle are located behind the insertion of the iris. Therefore, in the contracting of the muscle the circular fibers push the iris forward, tending to occlude the iridocorneal angle of the already shallow anterior chamber; this mechanism is aided by the pressure of the engorged lens and vitreous as well as by the increased size

of the lens due to age. In eyes with this condition there is a predisposition to glaucoma.

The same effect is obtained by dilation of the pupil in hypermetropic eyes. On the other hand, dilation has little influence on myopic eyes. Therefore, all other factors remaining equal, the hypermetropic eye is predisposed to glaucoma in a vastly increased proportion compared with the myopic eye.

Returning to a consideration of the modifications produced in the composition of the aqueous humor by alteration in the osmotic equi-

Fig. 5.—Vacuole formation due to edema of the cornea. Disappearance of Bowman's membrane.

librium, one notes that the proportion of colloids has been increased, and that albumin has transuded from the altered capillary walls which permit the escape of serum, as in cases of advanced arteriosclerosis in many of which retinal hemorrhages occur. Hemorrhagic glaucoma of this type is, in many cases, only an advanced degree of chronic simple glaucoma.

The changed aqueous humor, abnormally thick, finds some difficulty in filtering through the regulatory valves; this difficulty is increased not only in the spaces of the ligamentum pectinatum, but also by the generalized sclerosis of the iris stroma (fig. 4), which diminishes filtration to the suprachoroidal spaces.

Endophlebitic lesions, marked sclerosis of the structures surrounding the venae vorticosae and sclerosis of the vessels that connect Schlemm's canal with the episcleral veins aggravate still more the difficulty of aqueous filtration; they produce a condition similar to the colmatage filling-up process of Lagrange and a passive congestion on account of the impeded circulation in the venae vorticosae, thus considerably decreasing the functioning capacity of these safety valves. When such conditions persist, the nutrition of the eye decreases from day to day, and there is more transudation and edema.

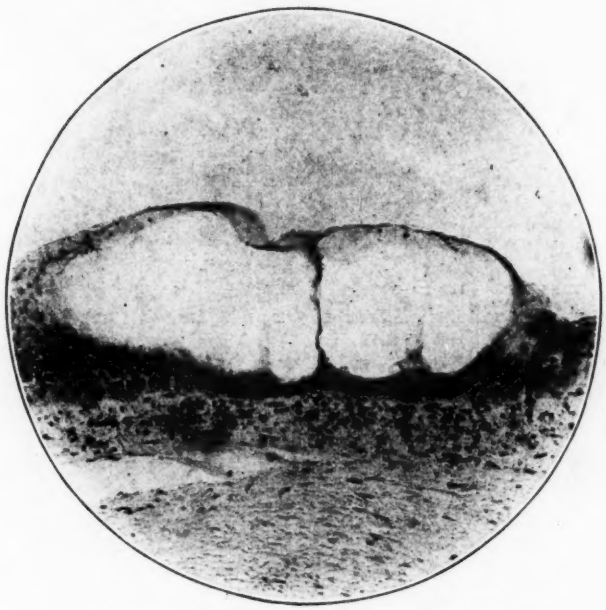

Fig. 6.—Large cyst formation in the epithelial layer of the cornea in a long-standing case of chronic simple glaucoma.

The interchange between blood plasma and tissue, and vice versa, becomes less and less. Toxic substances accumulate in the tissues, and the senile lesions become worse, owing to poorer nutrition, and a state of intra-ocular edema becomes established. The time finally arrives when, (1) by the combined action of the engorged vitreous and lens already increased in size by age, (2) by predisposition owing to anatomic conditions, (3) by contraction of the ciliary body pushing the lens forward, (4) by the thickened aqueous humor and (5) by the generalized sclerosis of the tissues, the periphery of the iris is pressed against the spaces of Fontana (hindering still more filtration in this safety valve) and, intra-ocular tension being increased, glaucoma is established.

Fig. 7.—A more advanced condition of edematous cornea; disappearance of Bowman's membrane which has been replaced by newly formed connective tissue (pannus degenerativus).

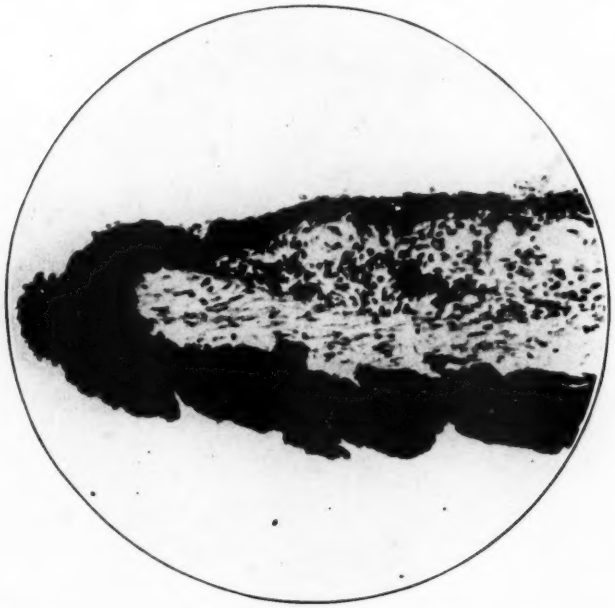

Fig. 8.—Ectropion uveae and emigration of pigment toward the anterior surface of the iris.

At first the iris only is in contact with the cornea; later, adhesions form; then a strong connective tissue deeply attaches the iris to the cornea, and the glaucoma becomes permanently established. The glaucoma parallels the advance of degeneration and atrophy in the different structures of the eye in addition to the hypertension, which of itself produces different lesions.

When the iris becomes adherent to the cornea, the symptoms become worse, and intra-ocular pressure increases still more. The aqueous humor is forced into the cornea, and the normal proportion of liquids

Fig. 9.—Marked atrophy of the ciliary body in a case of advanced chronic simple glaucoma.

and solids in this membrane is broken; the aqueous humor loses its equilibrium and transparency and becomes cloudy. The aqueous humor passes through the openings of the perforating nerves in Bowman's membrane, the epithelial layer becomes edematous, and small vacuoles are formed (fig. 5); in the later stages of this condition the vacuoles fuse, forming blisters and later cystic degeneration (fig. 6).

The superficial cells degenerate because of the poor nutrition and the trophic disturbances. Section of the nerve current occurs, owing to intra-ocular tension which presses the nerve in the suprachoroidal space against the sclera; the same nerves undergo degeneration in the edematous cornea.

The superficial epithelial cells keratinize, form vacuoles and fall, leaving irregularities on the surface of the cornea between the epithelial cells and substantia propria; a new connective tissue takes the place of Bowman's membrane, in some places forming what is called pannus degenerativus (fig. 7).

In the iris the circulation of the blood becomes difficult because of the constant elevation of the intra-ocular tension and the vascular obstruction due to modification of the structure of the vessels. Besides, the peripheral synechia strangles the root of the iris, with its vessels

Fig. 10.—The first stage of adhesion in peripheral synechia.

and nerves, by means of the proliferation of connective tissue. The cutting off of the blood supply produces atrophy of the iris stroma; the mesodermal part shrinks, and the ectodermal layer is exposed to the anterior surface, forming ectropion uveae (fig. 8).

The pigment of the ectodermal layer disintegrates and emigrates toward the anterior surface of the stroma of the iris, where it forms small granules (figs. 8 and 11); sometimes it is found in the posterior surface of the cornea.

The section of the nerve current paralyzes the sphincter of the pupil, which dilates. The irregular dilatation of the pupil and the disappearance of its circular form are due to an unequal degree of adhesion on the periphery and also to unequal sclerotic atrophy in the stroma of the iris.

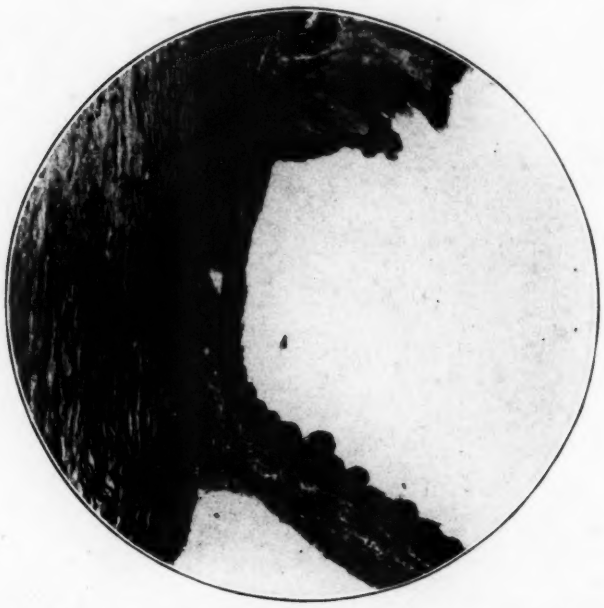

Fig. 11.—Advanced stage of peripheral synechia in chronic simple glaucoma, with atrophy of the iris and emigration of pigment toward the anterior surface of the iris.

Fig. 12.—Sclerosis of the central vessels and proliferation of the glia cells.

Fig. 13.—Beginning of glaucomatous cupping. Note the marked thickening of the central vessels.

Fig. 14.—Very deep glaucomatous cupping.

The ciliary muscle suffers the same disturbances from faulty nutrition and the progressive increase of tension which, stretching the ciliary nerves away from the sclera, cuts the nerve current, and the ciliary muscle ceases its work.

In this condition, the intra-ocular pressure finds a soft mass which yields easily, compressing the ciliary muscles (especially in hyperopia and emmetropia) against the rigid fibrous coat of the eye. In this compression, the ciliary muscles push the root of the iris against the periphery of the cornea and makes the possibility of reopening the iridocorneal angle more difficult.

Fig. 15.—Another instance of extreme glaucomatous cupping in chronic simple glaucoma.

In long-standing cases of glaucoma, the ciliary body and ciliary processes are considerably reduced, becoming very thin, as can be seen in figure 9.

At first, the root of the iris does not keep to the cornea; later, aided by the exuded albuminous liquid, adhesion of both structures takes place (fig. 10); still later, there is a proliferation of strong connective tissue between them (fig. 11).

The choroid suffers the same circulatory disturbances and atrophies; in advanced cases it becomes very thin. Pigment only is seen in its place. It is not unusual to observe a cataractous degeneration of the lens.

In the optic nerve the same sclerotic manifestations are seen as in the other membranes. As may be observed in figure 12, the vessels

Fig. 16.—Vascular degeneration in the fibers of the optic nerve near the lamina cribrosa.

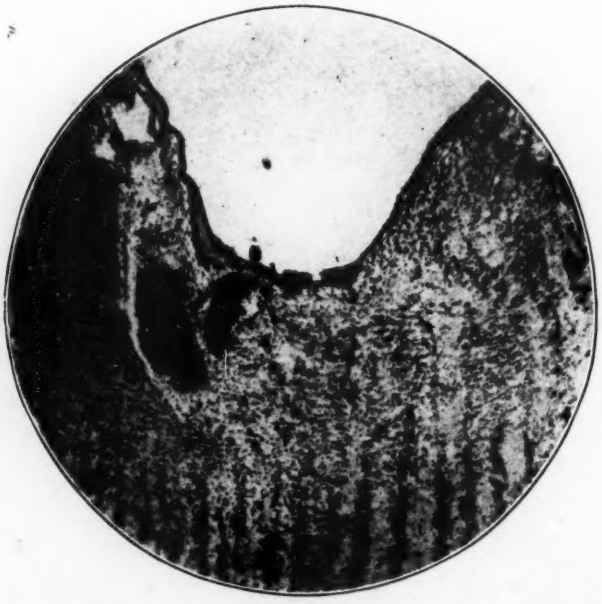

Fig. 17.—Thrombosis of the central vessels in glaucomatous cupping.

are surrounded by a dense connective tissue and glia. Partial cupping results (fig. 12), in which the resistant mass of vessels and connective tissue is conserved. Later, the intra-ocular tension pushes the lamina cribrosa backward, and the classic cupping of glaucoma is established (figs. 14 and 15).

Owing to the stretching of the fibers by the edge of the cupping, atrophy of the ganglion cells and fiber layers of the retina and poor nutrition, cystic degeneration of the tissues of the optic nerve takes place (fig. 16). Sometimes modification of the intima produces thrombosis in the central vessels (fig. 17).

Fig. 18.—Extensive cystic degeneration of the ora serrata and marked atrophy of the choroid.

In the retina, too, are found many changes due to the circulatory current, to stretching of the fibers and to intra-ocular pressure. In the ora serrata cystic degeneration is observed extending backward toward the posterior pole of the eye (fig. 18). This is due exclusively to poor nutrition and, in some cases, is the origin of hyaline deposits in this area (fig. 19) and throughout the whole region.

The ganglion cells and the fibers also participate in the general deterioration (fig. 20) consequent on the combined action of increased intra-ocular pressure, stretching of the fibers in the edges of the pathologic cup and poor nutrition. Cystic degeneration is observable in the external layers, owing to atrophy of the choroid which nourishes

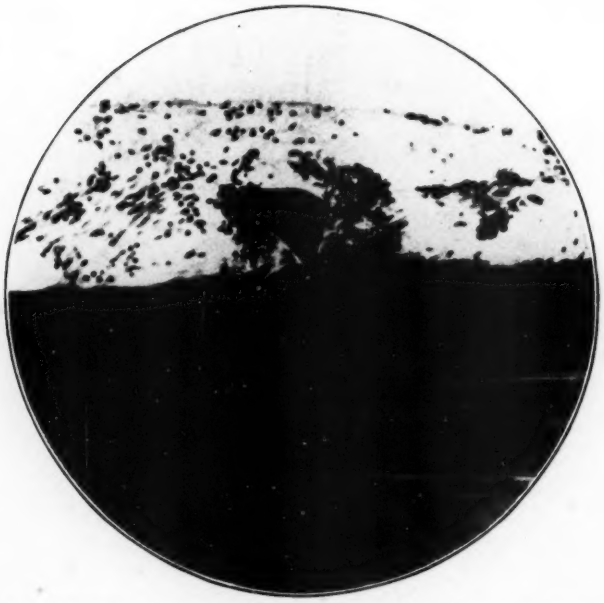

Fig. 19.—Hyaline degeneration (hyaline body) at the ora serrata.

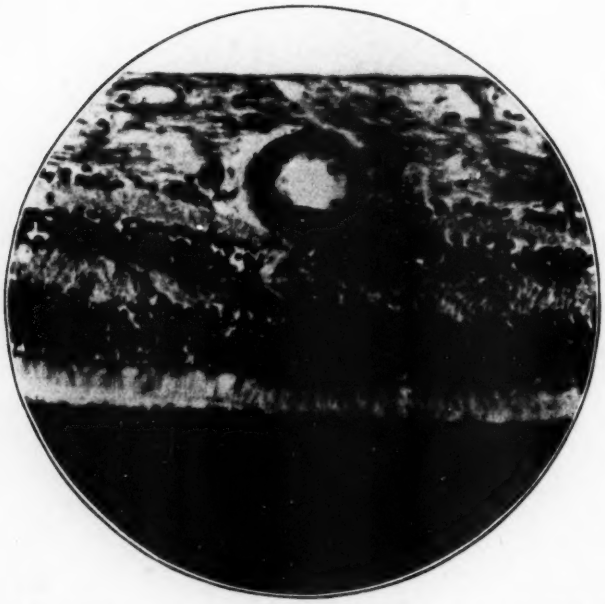

Fig. 20.—Atheromatous changes in a vessel of the retina and slight hemorrhages in between the outer granular and ganglion cell layers; marked atrophy of the ganglion cells in fiber layers.

them; this is easily seen in the region of the fovea centralis (fig. 21). Small hemorrhages can be found near the sclerotic vessels (fig. 20). Sometimes these hemorrhages are more extensive (fig. 22) and correspond not to different types of glaucoma, but to different degrees of the same process.

As can be seen in the example just described, this type might be called senile glaucoma, with the characteristic that the loss of equilibrium originates in vascular disturbances.

It should not be stated, however, that in glaucoma vascular disturbances are always present as the primary motivating mechanism or

Fig. 21.—Beginning cystic degeneration in the region of the macula in chronic simple glaucoma. Artefact detachment of the retina.

that the occurrence of glaucoma is always dependent on lesions of this sort. In cases of senile glaucoma this etiology seems to be clear, but there are other types of glaucoma in which one finds few or no pathologic changes that might explain the complex.

In the case referred to, the loss of equilibrium originates in local vascular disturbances, which may be considered a part of the changes in the whole organic vascular system, which are the natural result of generalized senile changes, subject to the laws of life. But it should be pointed out that there are many pathologic conditions that may produce such vascular disturbances as secondary manifestations. They

may be observed in metabolic difficulties, in diseases of the kidneys, in external and internal intoxications, in infectious diseases, especially syphilis, etc.

In all cases of this kind it is well known that glaucoma is the expression of a diseased organ in a diseased body, and that, of course, the entire system will be affected. There is a deficiency in the functioning of all the different organs, with a counterbalancing compensatory mechanism throughout the whole body. Here again one observes the complexity of biologic phenomena. One infers that the sufficiency of some organs tends to maintain the normal organic formula until the

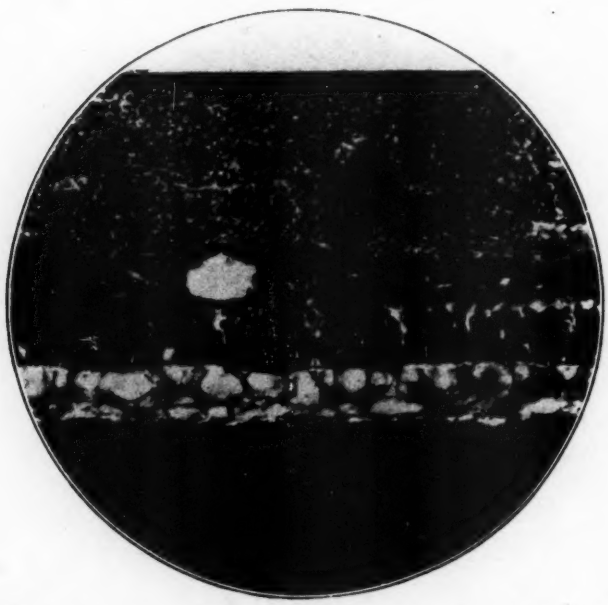

Fig. 22.—Extensive hemorrhage in ganglion cells and fiber layers of the retina. Cystic degeneration in the layers of the rods and cones.

equilibrium is broken and descendent, and that when a point is reached in this descent at which the formula cannot be reestablished by the organism itself without the influence of foreign intervention, the progressive premature lowering of all the organic complex proceeds until death intervenes. This explains presenile glaucoma.

Therefore, the outline to follow in cases of glaucoma is to discover, by careful examination, the deviation of the entire organism from the normal formula, from the normal standpoint of the whole; then to treat the local symptoms, which of themselves threaten the life of the organ, such as the hypertension of glaucoma, which leads to loss of vision.

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OCULAR TENDON TRANSPLANTATIONS

THREE NEW METHODS *

RODERIC O'CONNOR, M.D.
SAN FRANCISCO

The three new methods of ocular tendon transplantation make the new attachments external, in varying amounts, to the median vertical plane of the globe, with the idea of increasing the mechanical chances of securing outward rotation and of lessening tension on the portion transplanted. The illustrations show this mechanical aspect in a diagrammatic way.

The vertical recti will be used for ease of description, although the operations can be applied for isolated paralysis or paresis of any rectus muscle.

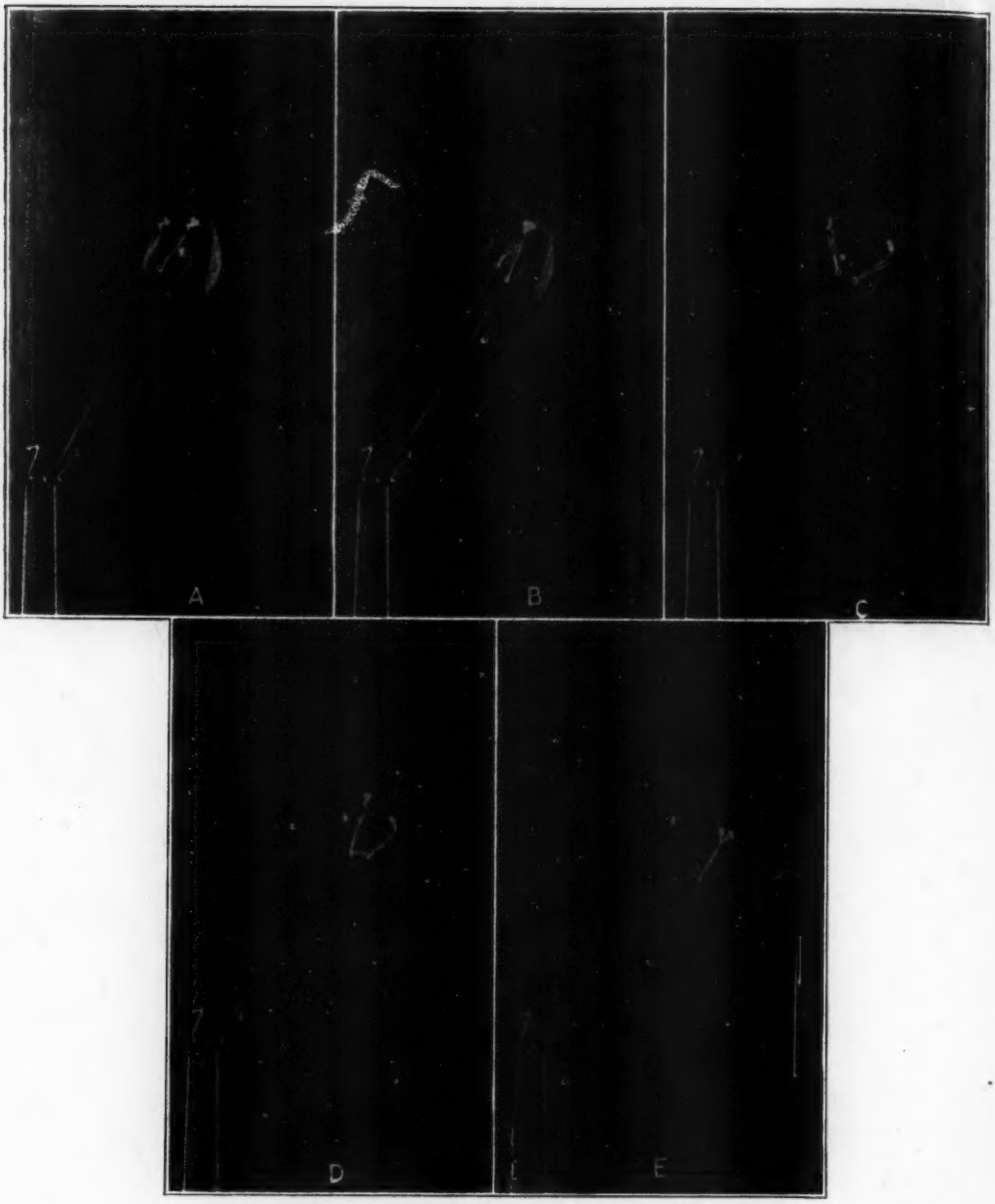
METHODS

First Method.—This method consists in a transplantation of the inner three fourths of the tendons of the vertical recti under the uncut outer fourth to the corresponding margins of the externus insertion. This operation was performed on both eyes of a patient of Dr. Hans and Dr. Otto Barkan at their request. The condition was double, complete, congenital abducens palsy in a girl 19 years of age.

This operation is far superior, mechanically at least, to transplantation of the outer halves for the following reasons: 1. All of the new attachment is far external to the median vertical plane of the globe, thus increasing the chance of securing outward rotation. 2. The transplants, if isolated far enough back, fall into their new places with practically no tension. 3. This happens because of the entire absence of an opponent and restraining effect such as occurs when the outer halves are transplanted and the inner half is left in place.

While the results were satisfactory in this patient, even to her, extremely rigid interni and a general orbital condition of ninteen years' duration had to be combated. Therefore, I am more than ever convinced that in such cases operation should be performed at the end of the first year, when the condition is congenital, or as soon as it is evident that recovery of function is hopeless, when the condition is acquired. As in all my work on muscles, I use my cinch-shortening loop in the transplant because it cannot pull out, especially when care is taken to leave the bunched fibrous insertion on the tendon by shaving it from the sclera. Otherwise, the technic is the same as in transplanta-

* Submitted for publication, Sept. 16, 1930.



A, a model with all the thread rubbers, representing the recti, set to balance the globe in the primary position. The superior rectus is divided to show the effect, in a mechanical way, of moving each half and both as described in the paper. In making these changes in the vertical recti no alterations in tension of the other rubbers were permitted. *B*, an outward shift of the pointer to about 7.5 degrees as the mechanical tendency of the third method. *C*, an outward rotation of 20 degrees as the mechanical tendency of shifting the outer halves of the vertical muscles. The restraining action of the inner half is clearly seen in contrast to the appearance in *D*. *D*, the inner half is moved to the externus attachment, and the pointer stands at 35 degrees. The freedom from tension is apparent. *E*, transplanting the entire muscle moves the pointer to 45 degrees. These mechanical effects can be increased by lessening tension on the internus (tenotomy or lengthening). The earlier the operation is done the better the chance of securing the desired result without tenotomy, which is important, as many of these patients have binocular vision, making it essential to preserve the power of convergence.

tion of the outer halves. It is remarkable how easily the transplants fall into their new places without tension.

Second Method.—This method consists in transplanting all of both vertical recti tendons. I have done this five times, the first case being reported in an appendix to a paper read at the Colorado Congress of Ophthalmology in 1924. Two operations resulted in marked vertical deviations, which led me to devise the first method; two gave satisfactory results and one a perfect result. This was a case of complete abducens palsy from fracture of the skull. Three years after operation, examination revealed vertical orthophoria, esophoria of 1 prism diopter and 18 degrees of outward rotation, and the patient claimed to be able to play tennis. When this method is used the tendons fall into place even more freely than with the first method, but no line of retreat is prepared (the "bridges are burned behind"), so I shall never, most likely, do this operation again.

Third Method.—In this method the inner half is cut free, applied over the outer half and there sutured. In several cases of abducens paresis, I have found this useful as an aid to full shortening of the externus. Similarly, in paretic interni the outer halves can be sutured over the inner.

OTHER THEORETICAL APPLICATIONS

1. In paralysis of the internus, transplant the outer halves of the vertical recti to the corresponding margins of the internus.

2. In paralysis of the inferior rectus, transplant the upper halves of the lateral recti.

3. In paralysis of the superior rectus, transplant the lower halves of the lateral recti. Practically, this might be an incorrect application because of a possible interference with downward rotation.

450 Sutter Building.

THE SYNERGISTIC ACTION OF ATROPINE AND EPINEPHRINE ON THE INTRINSIC MUSCLES OF THE EYE *

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AND
PETER C. KRONFELD, M.D.
CHICAGO

Up to about three years ago the conceptions of the physiology and pharmacology of the iris were comparatively simple. It was known that there were two muscles in the iris, the sphincter and the dilator, which were thought to act more or less independently of each other. The same assumptions were made for the influence of drugs on these two muscles. A group of drugs was known to influence only the sphincter, another group to act on the dilator. For the sphincter it was found that certain alkaloids had the power of increasing the tone, while certain others paralyzed it. A new point was brought out by Poos [1] when he was able to show active relaxation of the sphincter pupillae under the influence of cocaine or epinephrine, drugs which up to that time were thought to influence the dilator only. Poos studied the action of various drugs on the isolated sphincter and dilator. The former was prepared by excising about one half of the sphincter portion of a fresh animal or human eye. The excised portion was suspended between two fine clips in such a way that its length could be recorded on a kymograph. The whole specimen was submerged in physiologic solution of sodium chloride, to which the drugs were added subsequently.

With this method Poos was able to show active relaxation of the sphincter under the influence of cocaine, epinephrine and ephedrine. These drugs, which everywhere else in the body stimulate the sympathetic nerve, caused an active relaxation of the sphincter. This indicated strongly that the sphincter, like most of the smooth muscles, is under a double nervous control in which the parasympathetic is the motor nerve, while the sympathetic acts as the inhibitory nerve. It

* Submitted for publication, June 30, 1930.

* Read before the Chicago Ophthalmological Society, May 19, 1930.

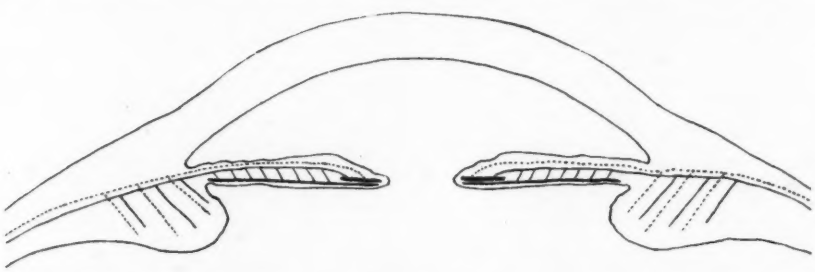
* From the Eye Clinic of the University of Chicago, Dr. E. V. L. Brown, director.

1. Poos, F.: Zur Frage der sympathischen Innervation des Ziliarmuskels, Klin. Monatsbl. f. Augenh. **80**:749, 1928.

was necessary, therefore, to change the conception of the innervation of the muscles of the iris. The sympathetic nerve, which has been known to control the dilator, is now thought to send some fibers to the sphincter (see accompanying illustration). Furthermore, Poos introduced the following new classification of the drugs that act on the iris.

1. Drugs with one point of action
   (*a*) Increased tone of the sphincter: pilocarpine, physostigmine, choline, insulin
   (*b*) Paralysis of the sphincter: atropine, scopolamine hydrobromide and their substitutes
2. Drugs with two points of action
   (*a*) Active relaxation of the sphincter and stimulation of the dilator: epinephrine, cocaine, ephedrine

From this classification it can be seen that there are two different groups of drugs with two different points of action which cause the



Innervation of the muscles of the iris. The solid line indicates the sympathetic nerve, and the dotted line the parasympathetic nerve.

same mechanical result on the iris. The epinephrine group and the atropine group both cause dilatation of the pupil. The combined action of the two groups is known as one of the best examples of synergistic action in ophthalmology. The action of a combination of atropine with cocaine, for instance, is much stronger than the added effects of the two components would be. It almost reaches the effect that would correspond to the product of the activities of the two components. Naito [2] studied this synergistic action on his own eyes. He first determined the threshold dose for the two single drugs and found two drops of atropine 1:100,000 or two drops of cocaine 1:50,000 just sufficient to cause a noticeable amount of dilatation of the pupil. In another experiment he used atropine 1:100,000 combined with cocaine, the amount of which always remained below the threshold for cocaine alone. The result was always a marked mydriasis, which corresponded to the effect of a much stronger solution of atropine than had been used.

2. Naito, I.: Ueber Kombinationswirkung von Atropin und Kokain am menschlichen Auge, Klin. Monatsbl. f. Augenh. **68**:154, 1923.

The synergistic action of atropine and cocaine is well known and is frequently used in the practice of ophthalmology. Especially in cases in which a maximum mydriasis is desired but not obtained by the use of atropine, a combination of atropine with cocaine should be tried. In patients up to the age of 45, atropine alone usually leads to a mydriasis that is sufficient for most practical purposes. However, if the physiologic tone of the sympathetic nerve is very low or the patient is elderly, the atropine leads to only a moderate mydriasis. In older people the resistance of the tissue of the iris and a beginning hyaline degeneration

TABLE 1.—*Results from Cocaine and Epinephrine Given After the Use of Atropine*

Patient	Age	Sex	Diagnosis*	Size of Pupil After 14 Drops of Atropine 1%, Mm.	Number of Drops of Cocaine 4% Every 10 Minutes	Time Between First Drop of Cocaine and Second Reading of Size of Pupil, Min.	Size of Pupil at Time of Second Reading, Mm.	Time After Subconjunctival Injection of 0.2 cc. of Epinephrine 1:1,000 at Time of Third Reading, Min.	Size of Pupil at Time of Third Reading, Mm.
E. W.	39	F	L C HY AS	6 Ver.† 5.5 Hor.	2	70	7		
R. R.	34	F	R C HY AS	6 Hor. 6.5 Ver.	6	60	7 Hor. 7.5 Ver.		
M. M.	21	F	R HY	7.5 Hor. 8 Ver.	6	60	8 Hor. 8.5 Ver.		
L. C.	31	M	L C HY AS	7	6	60	7.5		
S. D.	33	F	R MY	6	6	60	7.5	45	9
H. J.	17	M	L C HY AS	6	6	60	7.5	45	7.5 Ver. 8 Hor.
A. E.	34	F	R C HY AS	7	6	60	8	45	9
P. K.	19	M	L C HY AS	7	6	60	8	50	8.5
P. M.	44	M	L HY	6	6	60	7	50	8
O. S.	17	M	R C HY AS	7.5	..	..	..	50	8.5
A. M.	22	F	R C HY AS	7	..	..	..	45	8.5
E. L.	21	F	R C HY AS	7.5	..	..	..	45	9

* L indicates left; R, right; C HY AS, complete hyperopic astigmatism; HY, hyperopia, and MY, myopia.

† Ver. indicates vertical; Hor., horizontal.

of the sphincter portion of the iris prevent extensive changes of the size of the pupil if only the sphincter is paralyzed. In these cases it is necessary to add a stimulant for the dilator in order to get sufficient mydriasis. It can be shown in practically every case that a so-called complete atropine mydriasis can still be increased by the use of cocaine. In table 1, we present observations on a few cases in which, after the use of four drops of 1 per cent atropine a day for three days, various amounts of cocaine and epinephrine were given. Each of them showed an increase in the size of the pupil.

Especially for the examination of the extreme periphery of the fundus the combination of atropine with cocaine has been very satisfac-

tory. During the last two years the demand for as complete a mydriasis as possible has occurred in every case of retinal detachment in which one had to look for the retinal tears.

That atropine and epinephrine have a synergistic action on the iris is a well known and useful fact. Now the question arises: Can the same principle be applied to the ciliary body?

Before going into the study of this question one must be aware of the great difficulty encountered if one compares the action of certain drugs on the ciliary body with that on the iris. The size of the pupil represents solely the result of the action of the two muscles, and no other factor is involved, except the resistance of the tissues of the iris in elderly people. The only way of studying the action of drugs on the ciliary muscle is to observe the changes of the static refraction and of the range of accommodation that occur under the influence of these drugs. The changes of the refraction and the changes of the range of accommodation unfortunately represent not the actual amount of contraction of the ciliary muscle, but the changes of the curvature of the lens that occur under the influence of the contraction of the ciliary muscle. It must be expected that these two processes are not exactly parallel. So it is likely that the maximum possible amount of accommodation does not represent the maximum possible amount of contraction of the ciliary body, but only the extent to which the lens follows or yields to the contraction of the ciliary body. This consideration must be applied if changes of the range of accommodation after the use of drugs are interpreted. One must be aware of two possibilities, especially if the limits of accommodation are studied: (1) The limits represent the maximum or minimum contraction of the ciliary muscle, or (2) they indicate to what extent the lens can change the curvature of its surfaces. The upper limit or the maximum of accommodation is known to be determined by the ability of the lens to bend its surfaces. In early childhood this cannot be proved. But later, when the range of accommodation gradually decreases, it is interpreted as impaired ability of the lens to increase the curvature of its surfaces.

For the lower limit of accommodation or what is usually called relaxation of the ciliary body, it is hard to decide whether it indicates the maximum possible relaxation of the ciliary muscle or the maximum possible flattening of the surfaces of the lens. The following observations may throw some light on this problem.

For the pupil the effect of atropine is not the maximum that can be obtained. Therefore it was thought possible that the effect of atropine on the ciliary body did not represent the maximum relaxation. Cocaine and epinephrine are known to have a paretic influence on the ciliary muscle. Hamburger and Magitot denied that epinephrine had this influ-

ence, and Kaiser and Orr said that ephedrine did not have a paretic effect, but Poos was able to demonstrate this effect of both drugs in many experiments. These experiments support strongly the old theory of Morat-Doyon, according to which the sympathetic is the inhibitory nerve for the ciliary muscle. From this point of view it could be expected that cocaine or epinephrine could still increase the effect of atropine on the ciliary muscle. It was important to decide this question, mainly because atropine refractions are now a widely accepted routine procedure for the determination of static refraction. It would certainly change opinion about the atropine refraction as the best basis for the prescription of glasses if it could be shown that by a combination of atropine with cocaine a lower value for the refraction could be obtained. This has been the reason why the atropine refraction has become a routine procedure: It furnishes the lowest and most constant data.

TABLE 2.—*Changes of Static Refraction on the Instillation of Cocaine Following Complete Atropinization*

Patient	Age	Sex	First Refraction* Skiascopy Under Atropine	Second Refraction† Skiascopy Under Cocaine (+ Atropine)	Difference
S. D.	33	F	R +0.25	R +0.25	None
H. J.	17	M	L +1.00+1.50 cyl.×90	L +1.00+1.50 cyl.×90	None
A. E.	34	F	R +3.50+0.50 cyl.×90	R +3.75+0.50 cyl.×90	+0.25 sphere
P. K.	19	M	L +1.25+0.75 cyl.×90	L +1.50+0.75 cyl.×90	+0.25 sphere
P. M.	44	M	L +2.50	L +2.50+0.25 cyl.×180	None
M. M.	21	F	R +1.50	R +1.50	None
L. C.	31	M	L +1.25+0.75 cyl.×90	L +1.25+0.87 cyl.×90	Minus 0.12 cylinder

* First refraction under atropine 1 per cent, four times daily for three days.
† Second refraction under cocaine 4 per cent, one drop every ten minutes for six drops.

OBSERVATIONS

Our observations can be arranged in four groups: (1) refraction under cocaine after complete atropinization; (2) refraction under epinephrine after complete atropinization; (3) refraction under epinephrine and cocaine after complete atropinization, and (4) refraction under epinephrine compared with atropine refraction.

Table 2 shows the changes of static refraction as found by retinoscopy when, after complete atropinization (4 drops a day for three days, and 2 drops on the day of the examination, or altogether 10 mg. of atropine sulphate), 6 drops of 4 per cent cocaine were instilled, and the refraction was taken again. It is seen that the changes found lie within the limits of error. The same thing can be said about the changes that are found if atropine is combined with epinephrine (table 3) or with epinephrine and cocaine (table 4). One can conclude that the addition of cocaine or of epinephrine or of both drugs together does not change the effect of atropine on the static refraction to any notice-

able extent. This could be interpreted as an inability of the sympathomimetic drugs to add anything to the relaxation of the atropinized ciliary muscle or as an inability of the lens to flatten out any more than it does

TABLE 3.—*Changes of Static Refraction on the Injection of Epinephrine Following Complete Atropinization*

Patient	Age	Sex	First Refraction* Skiascopy Under Atropine	Second Refraction† Skiascopy Under Epinephrine (+ Atropine)	Difference
E. L.	21	F	R —2.75—0.50 cyl. ×15	R —2.75—0.50 cyl. ×15	None
A. M.	22	F	R +1.75+0.50 cyl. ×90	R +2.00+0.25 cyl. ×90	+0.25 sphere
O. S.	17	M	R +2.25+0.75 cyl. ×90	R +2.25+0.75 cyl. ×90	None

* First refraction under atropine 1 per cent four times daily for three days.
† Second refraction forty-five minutes after subconjunctival injection of 0.2 cc. of epinephrine 1:1,000.

TABLE 4.—*Changes of Static Refraction on the Instillation of Cocaine and Injection of Epinephrine Following Complete Atropinization*

Patient	Age	Sex	First Refraction* Skiascopy Under Atropine	Second Refraction† Skiascopy Under Cocaine and Epinephrine (+ Atropine)	Difference
H. J.	17	M	L +1.25+1.25 cyl. ×90	L +1.25+1.25 cyl. ×90	None
S. D.	33	F	R +0.25	R +0.25	None
A. E.	34	F	R +3.50+0.50 cyl. ×90	R +3.75+0.50 cyl. ×90	+0.25 sphere
P. K.	19	M	L +1.25+0.75 cyl. ×90	L +1.50+0.75 cyl. ×90	+0.25 sphere
P. M.	44	M	L +2.50	L +2.50+0.25 cyl. ×180	—0.25 cylinder

* First refraction under atropine 1 per cent four times daily for three days.
† Second refraction under cocaine 4 per cent, every ten minutes for six times followed by subconjunctival injection of 0.2 cc. of epinephrine 1:1,000.

TABLE 5.—*Effect of Epinephrine on the Static Refraction Before Atropinization*

Patient	Age	Sex	Previous Glasses	Manifest I	Refraction Under Epinephrine II	Refraction Under Atropine III	Difference III — II	Difference II — I
D. E.	17	F	None	+2.50	+2.75	+4.75 +0.25 cyl. × 90	+2.00 —0.25 cyl.	+0.25
M. B.	44	F	+0.50	+1.00 cyl. × 15	+1.00 cyl. × 15	+0.75 +1.00 cyl. × 15	+0.75	None
I. B.	34	F	—0.75	—0.50 —1.00 cyl. × 80	—0.50 —1.00 cyl. × 80	—0.50 —0.25 cyl. × 60	+0.75 cyl.	None
H. G.	34	M	—1.75 cyl. × 180	—1.75 cyl. × 180	—1.50 cyl. × 180	+0.25 —1.75 cyl. × 175	+0.25	—0.25 cyl.
			—1.25 cyl. × 170	—1.25 cyl. × 180	—1.25 cyl. × 180	—1.25 cyl. × 175	None	None

under atropine. In order to decide which of the two interpretations was correct, the effect of epinephrine on the static refraction was studied in a few cases before the atropine refraction was done. Table 5 shows the results obtained in four cases.

Case 1 was that of a girl, aged 17, who had never worn glasses. The first test performed was a manifest which revealed a hyperopia of 2.5 diopters in the left eye. On the same day the usual amount of epinephrine was injected, and the retinoscopy performed forty-five minutes later showed a hyperopia of 2.75 diopters. Then atropine was used, and the refraction taken under complete cycloplegia was plus 4.75 sphere with plus 0.25 cylinder, axis 90. From these observations one can conclude that the static refraction under epinephrine is very much like that found in an ordinary manifest; in other words, the influence of epinephrine on the tone of the ciliary muscle at the time of the most marked paresis of accommodation is little or nil. In the other three cases the observations furnish more evidence for this opinion. In case 2, a woman, aged 44, who had worn plus 0.5 sphere, readily accepted plus 1.0 cylinder, axis 15 in the first manifest. The following epinephrine refraction showed the same result, while when atropine was used the same cylinder combined with plus 0.75 sphere was found. In cases 3 and 4, in which almost the proper correction had been worn for some time, the manifest and the epinephrine refraction agreed also.

In a few other cases it was tried whether or not an injection of epinephrine would change the result of refraction obtained with homatropine hydrobromide, which is generally known as not leading to complete relaxation of the ciliary muscle. In none of these cases did the homatropine refraction show any change after the injection of epinephrine.

CONCLUSIONS

All these facts speak much in favor of the opinion that epinephrine, in the dose that was used in our cases, has no influence on the tonicity of the ciliary muscle and therefore no noticeable influence on the static refraction of the eye, although the range of accommodation is considerably reduced. Epinephrine and cocaine, therefore, are cycloplegics which prevent, or in smaller doses only reduce, the contractility of the ciliary muscle, but do not change its original natural tone. For atropine the observation still holds true that it causes the maximum possible relaxation of the ciliary muscle and so the minimum static refraction known up to date. The failure of the sympathomimetic drugs to increase the effect of atropine indicates strongly that the effect of the latter on the ciliary muscle cannot be increased by any method.

PRIMARY SARCOMA OF THE IRIS

REMOVAL BY IRIDECTOMY; NO RECURRENCE IN THREE AND A HALF YEARS *

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PHILADELPHIA

A boy, aged 10, was first seen on Nov. 6, 1926, with a history that four months previously a small pinkish growth had first been noticed on the colored part of the left eye. It had been slowly increasing in size. There was no history of injury or inflammation. The family history was unimportant.

In the lower outer sector of the iris of the left eye there was a flesh-colored tumor about 3 by 5 mm. It was ovoid and rested on the surface of the iris at the smaller circle and protruded into the pupillary space. The surface was smooth, and fine blood vessels were seen ramifying on the anterior surface of the growth. Seen with the biomicroscope the growth was somewhat translucent but not cystic. The surface was covered by ramifying vessels coming from the surface of the iris. The vessels on the iris were much larger and more tortuous than those on the growth. There was an irregular attachment of the tumor to the iris by means of several strands of pale pinkish vascular tissue. The iris reacted to light, with the exception of the sector on which the growth rested. No precipitates were present either on Descemet's membrane or on the anterior capsule of the lens. Vision was 5/5. There was a Mittendorf dot on the posterior surface of the lens. The fundus was normal. The Wassermann and Pirquet tests were negative.

A diagnosis of probable leukosarcoma of the iris was made, and as the case met the indications for removal by iridectomy this procedure was adopted. With the patient under ether anesthesia, as wide an incision as possible was made with a keratome, without touching the tumor, and the incision was enlarged with scissors. The iris was seized close to the growth and the mass brought outside the anterior chamber and removed, along with the iris tissue. The bleeding was so free that it was impossible to know at the time whether any of the mass was scraped off and left behind, as seemed possible since the growth was friable. After the hyphemia had disappeared, some detritus was seen in the anterior chamber. Again, with the patient under ether anesthesia, the posterior surface of the cornea and the lips of the wound were curetted.

After healing had taken place, the patient was placed under the care of Dr. George E. Pfahler for roentgen treatment. Dr. Pfahler's report was as follows: Treatments were given on December 7, 8, 13, 18 and 31, the patient receiving a total dose during this period of 175 per cent of an erythema dose. For these treatments low voltage technic was employed, corresponding to a 9 inch spark gap, 5 milliamperes at 30 cm. distance and 6 mm. of aluminum filter. He also received high voltage roentgen treatment over the temporal zygomatic regions and the left cervical region. Over this area he was treated on Dec. 9, 10, 11, 14 and 16, 1926, and Feb. 8, 9 and 10, 1927, receiving a total of 215 per cent of an erythema dose, with the use of 200 kilovolts at 50 cm. distance filtered

* Submitted for publication, Sept. 20, 1930.

* Presented at the Meeting of the American Ophthalmological Society, Hot Springs, Va., June 3, 1930.

through a 0.5 mm. of copper and 1 mm. of aluminum. He had received no treatment since that date. There was no loss of eyebrows or lashes, nor any damage to the skin.

The tissue was examined by Dr. P. DeLong, who reported the growth to be a leukosarcoma.

For the purpose of this report, at Dr. DeLong's request, a detailed description of the histology was made by Dr. Joseph McFarland. As a result of this study, Dr. McFarland was of the opinion that the tumor was a sarcoma. In support of this view, he found the following evidence:

1. The tumor was almost entirely composed of cells of an elongated spindle shape with ovoid nuclei. No definite substance of any kind could be made out between these cells.

2. The blood vessels were large, but of such extreme simplicity of structure that their walls consisted only of endothelium.

3. Although the cells at first glance appeared to be extremely uniform, regularly gathered into bundles that curled this way and that, more careful examination showed that many of the nuclei were swollen or shrunken, and some were greatly deformed.

The patient was last seen on April 11, 1930. He was in good health. The eye presented the following condition: vision with -2.50 sphere $= +3.00$ cylinder axis 75 was 6/20. There was an operative coloboma of the iris from 2 to 5 o'clock and a small triangular opacity in the deeper layers of the cornea extending from the peripheral scar of the incision a few millimeters into the cornea. There was a similarly placed irregular opacity in the capsule of the lens immediately beneath the corneal opacity. There was a slight increase in the diffuse granular opacities throughout the posterior cortex of the lens, first noticed in February, 1929.

In 1918, S. Gifford treated a patient who had a leukosarcoma with radium, and although the growth diminished under the treatment, the pain induced by its application compelled enucleation.

Recently, R. Foster Moore resorted to the insertion of radon seeds into a sarcoma of the choroid, because the patient's fellow eye was blind, and he refused to have the afflicted eye removed. The radon seed used was of 1 millicurie strength and was filtered through 0.5 mm. of platinum. A conjunctival flap was reflected; an incision was made in the sclerotic just large enough to admit the seed; the knife was then thrust into the globe, and the seed inserted directly into the thickest part of the growth so that if possible it would be completely embedded in it. It was removed at the end of fourteen days.

As some shrinkage of the growth was noted, a second seed of 5 millicuries was inserted four months later. One year after the first treatment, the growth had become so shrunken that it was seen with difficulty. It was a dull ashen gray.

In 1902, Wood and Pusey compiled ninety cases of primary sarcoma of the iris. A search of the literature since that period reveals about sixty-five additional cases, of which sixteen were leukosarcoma. In fifteen of the sixty-five the growth was removed by iridectomy. There

A B

Fig. 1.—*A*, leukosarcoma of the iris. *B*, leukosarcoma of the iris; appearance with biomicroscope.

were but two recurrences after a period of from five months to sixteen years after the operation.

Notwithstanding these results, the concensus is that excision of the globe is the conservative treatment. Axenfeld considered iridectomy permissible when certain conditions exist; these conditions have been elaborated by Duke-Elder to be: a small neoplasm, well defined, situated

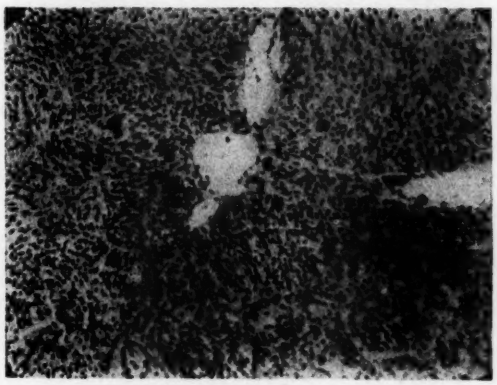

Fig. 2.—Histopathology of growth; low power.

Fig. 3.—Histopathology of growth; high power.

at or near the pupillary margin of the iris and not encroaching on the ciliary border of this tissue; normal intra-ocular tension and good vision in the affected eye; facilities for complete removal provided by iridectomy.

While there can be no difference of opinion as to the truth of the dictum that excision of the eye is the safest procedure in the presence of intra-ocular malignant neoplasm, in the light of present knowledge and experience of the value of roentgen and radium in the treatment

of growths, together with the fact that surgical removal alone of sarcoma of the iris has resulted in a cure, may one not hope that by following up the surgical treatment with the application of x-rays or radium, as was done in the foregoing case, a functioning eye may be preserved in a larger number of these cases?

The case which has been reported was referred by Dr. Calvin C. Rush, and the excellent water-color sketch was made by Dr. James T. Shipman.

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SENSITIVE METHODS FOR THE DETECTION OF BJERRUM AND OTHER SCOTOMAS*

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In a former paper two of the present writers described methods for increasing the diagnostic sensitivity of perimetry with the form field stimulus. It was pointed out that the methods described should be especially effective and feasible in that branch of field study that is commonly called scotometry. It is our purpose in the present paper to show with what success the methods may be used in the early detection of the Bjerrum and other scotomas.

In the practice of scotometry with the form field stimulus, the means for increasing sensitivity are comparatively obvious. In order to pick up faint or incipient scotomas, it is necessary only to decrease by sufficient amount the visibility of the stimulus.

The amount of increase in sensitivity that can be secured in this way clearly is limited only by the threshold of visibility. The three most important factors in the visibility of an object are its size, its difference from the background and the intensity of the illumination. The influence of the first two of these needs no comment. The influence of the third probably merits a word of explanation. The visual or sensation difference between an object and its background for a given difference in reflecting power increases rapidly with increase in the intensity of the illumination received. Objects become more easily distinguished from their background, therefore, as the intensity of the illumination is increased. The increase is greater in case of white objects on black or light objects on dark backgrounds than in case of black objects on white or dark objects on light backgrounds. Also the amount of increase varies with the state of adaptation of the eye. The decrease in visibility of the test object that is needed to secure greater sensitivity in scotometry may be produced, therefore, by decreasing the size of the object, by decreasing the difference in coefficient of reflection between object and background, by

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decreasing the intensity of illumination and by the combination of any two or all three of these procedures.

Great sensitivity can be added to scotometry also by the use of colored stimuli. Two reasons may be suggested for this. 1. The achromatic sensitivity of the eye is enormously greater than the chromatic sensitivity. The use of color stimuli, therefore, is roughly analogous to the use of form stimuli of low visibility. In the accompanying table is given a comparison of the achromatic and chromatic sensitivity of the eye to spectrum lights in different parts of the retina, central and peripheral.[1] 2. The color processes may have an especial susceptibility to pathologic conditions.

Ratio of Achromatic to Chromatic Sensitivity to Spectrum Light at Different Degrees of Excentricity, Dark Adaptation *

Degree of Excentricity in Field of Vision	Temporal 0°			Nasal 180°			Upper 90°			Lower 270°		
	Red (673 mμ)	Green (522 mμ)	Blue (468 mμ)	Red (673 mμ)	Green (522 mμ)	Blue (468 mμ)	Red (673 mμ)	Green (522 mμ)	Blue (468 mμ)	Red (673 mμ)	Green (522 mμ)	Blue (468 mμ)
0	1.1	2.0	2.3	1.1	2.0	2.3	1.1	2.0	2.3	1.1	2.0	2.3
5	12.3	21.2	11.3	3.7	27.8	23.2	8.2	36.1	11.1	7.6	40.7	12.8
10	18.7	48.2	13.3	4.3	58.1	25.6	37.8	114.2	8.0	8.9	93.6	13.0
15	Blind spot			8.7	194.2	179.6	24.3	200.2	10.2	34.9	1291.3	40.8
20	11.7	1686.5	40.5	23.0	965.0	208.7	20.7	1022.3	22.4	32.9	2460.0	181.8
30	635.7	5090.4	221.1	85.8	5081.3	514.4	801.4	3680.4	42.3	172.9	5652.5	589.5
35	....	....	....	....	....	....	729.5	....	....	....	....	....
40	2542.0	....	467.1	168.1	14163.5	....	....	....	....	228.3	11721.5	....
45	....	....	....	....	....	....	1083.2	....	....	....	....	....
50	3322.6	....	....	429.1	....	....	....	....	....	220.3	....	....
55	....	....	....	....	....	....	1430.3	....	....	....	....	....
60	4014.3	....	....	252.1	....	....	....	....	....	1459.0	....	....
70	5224.5	....	....	....	....	....	....	....	....	1709.0	....	....
80	6871.8	....	....	....	....	....	....	....	....	8521.1	....	....
90	2801.1	....	....	....	....	....	....	....	....	....	....	....

* The visual angle of stimulus was 1 degree. The comparisons made in this table are, it will be noted, for results obtained with spectrum lights. Data are not available as yet for a comparison of the achromatic sensitivity to colorless or white light with the chromatic sensitivity to colored light. Also the comparisons are for dark adaptation. For light adaptation, such large ratios would not be expected. In dark adaptation, too, the extent and distribution of chromatic sensitivity are not the same as in light adaptation. For example, according to the results obtained in the foregoing study, sensitivity to blue and green does not extend beyond 60 degrees in any meridian for the dark-adapted eye.

In order to test this point, it will have to be determined whether color stimuli still have a greater diagnostic sensitivity than form field stimuli when both are equalized in visibility for the normal eye.

In these attempts to increase the diagnostic sensitivity of the functional tests of the eye, a part of our incentive is to render them useful for general as well as for ocular diagnosis. Examination of the functions of the eye offers special advantages along these lines for the following reasons. (*a*) The retina is the most sensitive recorder in the body. The maximum sensitivity of the eye is approximately twice that of the ear, for example. It is thus extremely susceptible to every sort of change

1. These results are taken from a study made by H. A. Wentworth under the direction of two of us (Ferree and Rand).

and disturbing influence. (*b*) The ramifications of its conducting fibers are extensive and diffuse, and its sympathetic connections are important. (*c*) The location of the percipient elements of the retina between two networks of blood vessels makes them extremely susceptible to any influence that produces change in either the blood or the circulation. Also, less directly, the network of blood vessels around the zonule subjects the retina to the influence of changes in the circulation. (*d*) More is known about the functions of the eye than of any organ of the body. (*e*) More reliable and elaborate methods and apparatus have been devised for studying and testing these functions than have been devised for any other organ of the body. Because of the supreme importance of the eye to all observational and experimental work, many sciences have contributed extensively to its study and examination.

In this connection, it may be said further that as an aid to diagnosis the functional test has the following advantages as compared with the objective methods of examination of the eye:

(*a*) As a rule, or in the greater number of cases at least, functional disturbances precede detectable structural changes in the fundus in the development of pathologic conditions. An earlier diagnosis is thus rendered probable by the subjective method of examination. The hope of successful treatment rests on an early diagnosis.

(*b*) A more minute search can be made by the subjective than by the objective methods of examination. It is obvious that the minuteness of examination by the subjective methods is limited only by the threshold of space discrimination.

(*c*) Pathologic disturbances posterior to the fundus of a kind or of a magnitude that do not produce a visible change of structure in the fundus cannot be detected by the objective methods of examination. It is probable that many of these disturbances can be detected by the subjective methods at an early stage in their development, and that all of them can be detected at some stage.

(*d*) Results by the subjective methods of examination can be subjected more or less satisfactorily to numerical treatment and comparison.

(*e*) The objective method of examination deals with a static, the functional test with a dynamic, condition— a dynamic condition of the entire sensorium. The functional tests in proportion to their comprehensiveness and sensitivity scrutinize minutely the sensorium in action, and there is little in the way of depression or disturbance that should escape their survey.

In making these comments, however, there is of course no thought that either method can be substituted for the other. Each method tells a story of its own, and each has important details, both supplementary

and corroborative, to add to the diagnostic picture. The goal to be striven for is that each shall be given its highest development and efficiency. To this end attention should be paid at this time to the great possibilities that are offered by the subjective methods, particularly with reference to their range of application, high sensitivity and the thoroughness with which the study can be made and the results treated and analyzed.

METHOD OF WORKING

The cases were studied by means of the tangent screen of the Ferree-Rand perimeter. It has been pointed out in previous papers that in using the tangent screen of this instrument all the precision of control of variable factors may be attained that is had in the determination of the limits of the field on the arc of the perimeter. The delicacy and exactness of determination that are thus made possible are absolutely necessary to secure such results as are to be presented in this paper. The introduction of features to add to the diagnostic sensitivity would be of little avail if their action were to be masked and confused by the influence of factors that are not pathologic; further, little sureness could be felt with regard to prognosis if methods and appliances were not used that are known to give constancy of result from sitting to sitting when pathologic changes are not present. When such conditions of working are used and we find, for example, that during the incipient stages of the development of a Bjerrum scotoma it is sometimes present and sometimes absent, or that it disappears under the influence of a miotic or other treatment, we feel justified in believing that we are actually observing a function in process of change, and not just another vagary of technic.

The tangent screen was adjusted to a distance of 33 cm., and the appropriate fixation device was inserted. To insure constancy of the illumination of the stimulus and its surroundings, the arc of the perimeter was turned into the meridian that was being tested. The test objects mounted on the light rods designed for use with the tangent screen were used as stimuli. Whenever possible, the direction of movement of the stimulus was from the blind to the seeing region. Alternatives were (1) moving the stimulus in both directions and averaging the results, and (2) moving the stimulus from the sensitive to the blind area. The first of these possibilities was decided against for the following reasons: (*a*) In preliminary trials the stimulus was moved in both directions in order to determine whether for a given subject there was an appreciable difference in result. In nearly every case difference, when present, was found to be due largely, if not entirely, to the slowness of the subject's reaction in reporting the appearance or disap-

pearance of the stimulus. In proof of this, it was found that the two sets of results could be made to agree closely when due care was taken to regulate the speed of movement to correspond as closely as possible with the speed of the subject's reaction. (*b*) The movement in one direction allowed a greater number of points to be determined without fatiguing the subject.

The second of the aforementioned possibilities was decided against because experimentation showed that more reproducible results can be obtained when the stimulus is moved from the blind to the sensitive area than when the movement is in the opposite direction. Two reasons may be assigned for this. (*a*) A better control of fixation is possible. There is a tendency for the eye to follow the movement of the stimulus when it is in the field of view. (*b*) Owing to fatigue and adaptation, the stimuli, especially when colored, do not disappear as sharply when passing into the blind area, as they make their appearance when emerging from this area.

Frequent checking was, of course, resorted to when the determination, as indicated by undue or unusual irregularity of outline of the scotoma, seemed to demand it. Great care was taken, however, not to fatigue the eye unnecessarily. When there was doubt of the accuracy of the subject's report, a blank was given; i.e., the stimulus rod was turned over and the reverse side of the target presented. This check is needed frequently with suggestible subjects. Whenever possible, the direction of the movement of the stimulus was along a line at right angles to the outline of the scotoma at the point under investigation. In the preliminary search for the Bjerrum scotoma, the movement was along the radius of the tangent screen from a peripheral point toward the center, through the area of susceptibility. The subject was required to report either loss or any noticeable dimming of sensation. When a scotomatous or suspicious area was found, a procedure had to be adopted to suit the individual case. It is obvious that the technic needed to verify the indication and to outline the scotomatous area as to detail cannot be described in general terms. The examiner must show versatility and have at his command an ever enlarging stock of methods, devices and materials, and the ability to improvise. Each case is a bit of research and a stimulus to new achievement.

The form stimuli employed were 1, 0.5 and 0.17 degrees white and 0.5 degree gray viewed against the black tangent screen as background. At the distance of the tangent screen (33 cm.) the sizes of these stimuli were 5.8, 2.9 and 1 mm., respectively. The coefficient of reflection for the white stimuli was 78 per cent, for the gray stimulus 19 per cent (approximately midgray) and for the black background 3.5 per cent. The color stimuli used were 1 and 0.5 degrees blue and red and 1 degree

green [2] pigments of the Heidelberg series. The blue and red stimuli were used with the dark gray tangent screen supplied with the perimeter, coefficient of reflection 9.6 per cent, and the green with the light gray screen, coefficient of reflection 27 per cent.

It is important for precise and sensitive scotometry that the background against which a colored stimulus is seen should be of the same brightness as the color. In this way alone can the troublesome factors of preexposure and physiologic induction or contrast be eliminated, and the possibility avoided of the observer's reporting the stimulus as visible when it is seen as white or a light gray, which would be the case if a black background were used; or as black or a dark gray, if a light background were used. The customary use of a black tangent screen for all stimuli, form and color, is doubtless to a great extent responsible for the limited advantage that has been taken of the great sensitivity that color shows for the detection of scotomas. The results to be presented in this paper will demonstrate, we believe, that colored stimuli far surpass form stimuli in this respect. With a gray background of the correct brightness, the determinations, moreover, are quick and easy to make, and have a satisfactory reproducibility.

For the sake of comparison, it is desirable that all of the stimuli provided should have been used in each case. With clinic cases this is not always possible. The patient cannot always be secured for an adequate length of time at one sitting, and frequently a return sitting cannot be arranged. Because of these difficulties, a great deal of our data, though instructive, cannot be used for publication, and the comparisons for the cases presented are not always as complete as we should like to have them.

Since neither diagnosis nor the presentation of cases is the purpose of this paper, space will not be taken for the inclusion of all the medical and clinical data that are available for each case. We are interested only in showing the comparative sizes of the scotomas that were obtained with the different stimuli and in determining with which stimuli the detection can be made earliest in the development of the condition and with which the smallest change in the advance or recession of the condition can be picked up.

RESULTS

The results selected for presentation are shown in charts 1 to 17. They are typical of the results obtained with the stimuli employed,

2. A green stimulus smaller than 1 degree has not been found to be generally practicable because the area sensitive to it is not sufficiently large. For many eyes, having no pathologic change the field for 0.5 degree green, for example, lies inside the blind spot. Even with 1 degree green, this sometimes occurs, but not often.

covering a much larger number of cases. As was stated in the preceding section, only a brief presentation of clinic data will be made for each case, summarized from the data available.[3]

The results of the examinations with the tangent screen are presented in the form of charts in which the blind spot and scotomas as determined with the different stimuli are mapped. In making these maps for the different stimuli, the following scheme was used:

Areas blind to form are filled in solid with black, dark gray or light gray, the darker background being used for the stimulus showing the least sensitivity in each chart. Areas in which the form stimuli are seen dimly are outlined but not filled in, with the exception of two cases (charts 15 and 17) in which a stimulus of high visibility was seen dim over part of an area blind to a stimulus of lower visibility. This area blind to one stimulus and faintly sensitive to the other is filled in with a checker-board pattern. Areas blind to color are filled in with light gray, and black lines are drawn over the gray background as follows: for blue, diagonals running from upper right to lower left; for red, diagonals running from upper left to lower right; for green, dotted diagonal lines running from upper right to lower left. Areas in which the colored stimuli are seen dim are filled in with the diagonal lines selected for the color in question, but on a white not a gray background.

Both incipient and more advanced cases have been selected for presentation: incipient cases to show the value of the sensitized methods for the detection of a pathologic condition, and the advanced cases to show how much more delicately the actual extension of the condition can be detected with the sensitized methods.

CASE 1 (charts 1 and 2).—Mrs. M. C., aged 60, had bilateral chronic glaucoma simplex. She had been under observation since July, 1928. At that time pilocarpine was used because the tension in the left eye was slightly above normal. Since then, treatment with pilocarpine had been continued as needed in both eyes. Under the miotic, the tension varied between 25 and 32 [4] and was frequently higher in the left than in the right eye. Pain resulted if the pilocarpine was discontinued. The disks were not glaucomatous. The vision was 20/15 in both eyes with +3.00 spheres, with +2.75 spheres added for near vision.

The blind spot for the right eye of this patient was mapped on Jan. 7, 1930, and the test made for scotomas. The stimuli employed were 0.5 degree white on black, 0.17 degree white on black, 1 degree green, 0.5 degree red and 0.5 degree blue. The background for the colored

3. These data were taken from the records of the Wilmer Institute. Dr. W. H. Wilmer and the members of his clinical staff gave us their assistance.

4. All values of intra-ocular pressure are given in terms of the revised Schiötz scale.

stimuli was in each case of gray of the brightness of the color. The area of the blind spot and scotoma as obtained on the tangent screen was measured with a planimeter. The results in square centimeters are given also in chart 1. Inspection of the maps and data given in chart 1 shows a marked enlargement of the blind spot for the blue and red stimuli with scotomatous extensions both above and below, both features being much more pronounced for the blue than for the red stimulus. For both stimuli in this case the scotoma was continuous with the blind spot. There was perhaps also a slight enlargement for 1 degree green. The other stimuli showed nothing of diagnostic significance.

In chart 2 are given maps for the left eye of this patient from results obtained on different dates. The purpose was to make

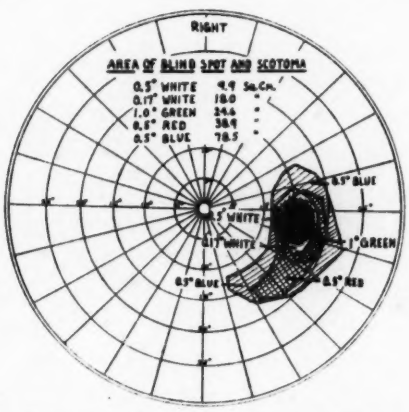

Chart 1 (case 1).—Bilateral chronic glaucoma simplex. The blind spot and scotomatous extensions when mapped with stimuli of different diagnostic sensitivity.

a comparison of the sensitivity of the different stimuli for the detection of changes in the condition. The stimuli used were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 1 degree red, 1 degree blue, 0.5 degree red and 0.5 degree blue. The tests were made on Dec. 21, 1929, and on Jan. 7, Jan. 24, Jan. 27, May 23, May 27 and May 30, 1930. From December 21 to May 27, the results for the form stimuli show no significant change. The results for the colored stimuli show enormous changes. These changes are shown in sequence in chart 2, *A*, *B*, *C*, *D* and *E*. On May 23, the first detection of change was made with the form stimuli.

The examination made with the tangent screen on December 21, did not reveal any enlargement of the blind spot with any of the stimuli employed. An examination made with the more accurate blind spot screen, designed to be inserted in the stimulus carriage of the perimeter,

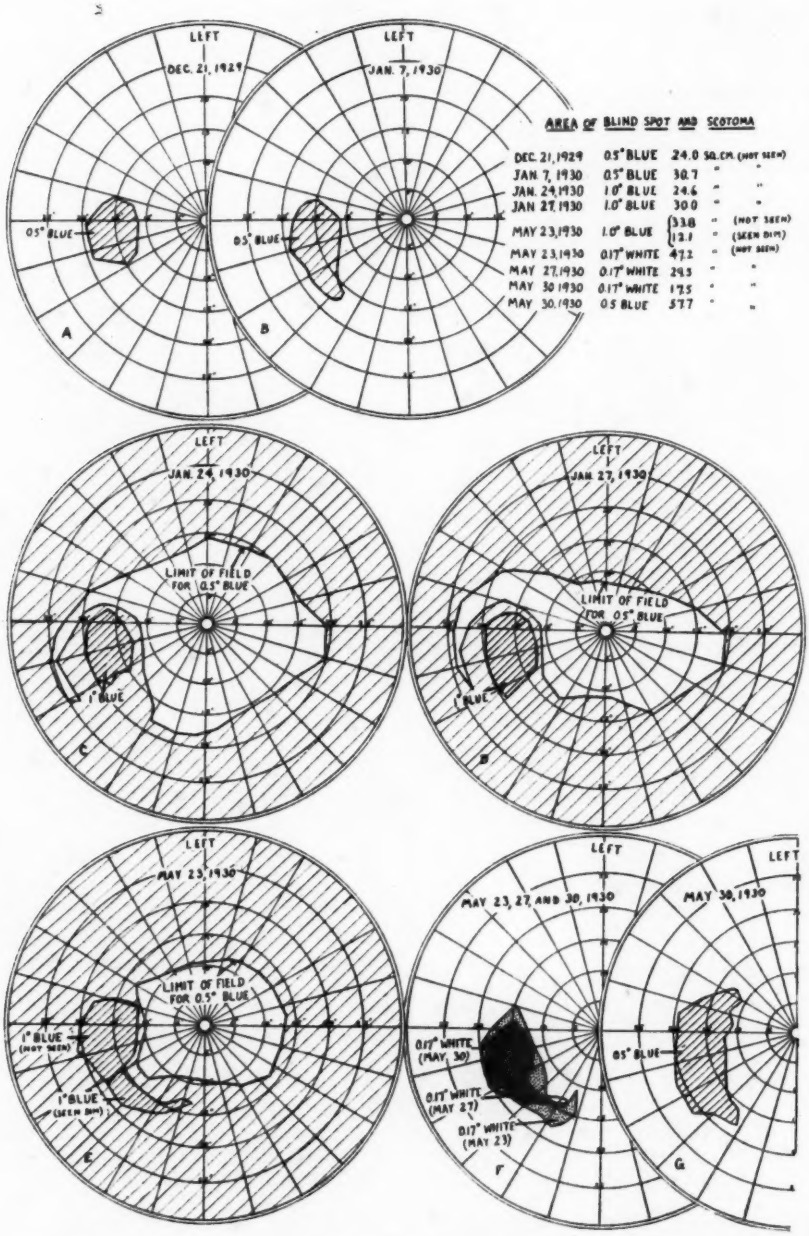

Chart 2 (case 1).—A comparison of the sensitivity of different stimuli for detecting changes in the size and shape of a scotomatous enlargement of the blind spot over a period of time.

showed, however, an enlargement for 0.5 degree blue, but for none of the other stimuli. On this date the tension was 24. A study of the fields showed contraction for 1 degree white on black (average breadth in the eight meridional quadrants, 55 degrees) and a nasal step of 12 degrees; for 1 degree blue, a field borderline in size (average breadth in the eight meridional quadrants, 33.9 degrees) and a nasal step of 6 degrees.[5] On January 7, on the tangent screen there was no enlargement of the blind spot for any of the form stimuli; there was enlargement for 0.5 degree red and 0.5 degree blue, however, with scotomatous extensions below. The tension on this date was 30. On January 24, the blind spots for 0.5 degree red and 0.5 degree blue were greatly enlarged, with scotomatous extensions below which merged with the blind area produced by the contracting field. The limits of the field of sensitivity for these stimuli fell to the nasal side of the blind spot, but above the blind spot a narrow, sensitive strip remained, which extended out and down in an irregular arc nearly encircling the blind spot. For 1 degree red and 1 degree blue, the blind spots were somewhat enlarged. For all the form stimuli, the blind spot was normal. The tension on this date was 30. On January 27, for 1 degree blue, the blind spot showed considerably greater enlargement than on January 24, and for 0.5 degree blue the residual sensitive area had contracted still further. The red stimuli were not used on this date. The blind spot for all the form stimuli was still normal. The tension was 30. A study of the fields made on this date showed no significant change in size for 1 degree white; the field for 1 degree blue, however, had contracted well below the

5. This, it will be noted, is an instance in which the disturbance in function could be detected by perimetry at a time at which it could not be detected by scotometry in the paracentral field. The case also serves as an illustration that the form stimulus may show a greater diagnostic sensitivity than color stimuli when the diagnosis is based on size of field. As bearing on the explanation of both of these points, it may be pointed out that in the early stage of the development of the condition the far periphery of the retina alone may have been affected. Of the stimuli employed, the 1 degree white is the only one with which this part of the retina could be examined. To have tested it for color sensitivity larger stimuli would have had to be employed. In explanation of the second of the points, attention may be called also to the large range of scatter of results obtained with color stimuli because of their greater sensitivity for picking up individual differences. This larger range of scatter reduces the diagnostic sensitivity of color stimuli when the diagnosis is based on size of field because of the tendency for the results for normal eyes to overlap those for pathologic eyes. It is possible that the blue field would have shown greater contraction than the form field could the comparison have been made in both cases with fields which were taken before pathologic changes have set in. It is because it is rarely possible to make such a comparison in the individual case with fields obtained prior to the suspicion of pathologic changes that we have to resort in diagnosis to such devices as range of scatter in a sample group.

borderline (average breadth in the eight meridional quadrants for 1 degree white, 53.3 degrees; for 1 degree blue, 30.3 degrees). On May 23, the examination was made after a slight attack of pain and blurred vision during the previous night. For 0.17 degree white, there was enlargement of the blind spot with a scotomatous extension below. This was the first time during the series of tests that an indication of change of condition was obtained with a form stimulus. For 0.5 degree white and 0.5 degree gray, the blind spot was normal. For 1 degree blue, the blind spot showed further enlargement as compared with January 27, and there was a pronounced scotomatous extension below in which the stimulus was seen dim. For 0.5 degree blue, the sensitive area had contracted within the 15 degree isopter and at places had reached the 10 degree isopter. The tension was 22½. A brief examination on May 27 showed an improvement in the condition. For 0.17 degree white the scotomatous extension below the blind spot was considerably smaller than on May 23. No examination was made with the colored stimuli. The tension on this date was 26. On May 28, the patient was admitted to the hospital and given intensive treatment.

On May 30, an examination was made again with both the tangent screen and the perimeter. A marked improvement was shown. The blind spot was normal for all the form stimuli, and for the first time since January 7 it could be mapped with 0.5 degree blue and 0.5 degree red. For both of these stimuli an enlargement was shown, and with 0.5 degree blue there were small scotomatous extensions both above and below. The fields on this date for 1 degree white and 1 degree blue as determined on the perimeter were within the normal range (average breadth in the eight meridional quadrants for 1 degree white, 64.6 degrees; for 1 degree blue, 40.3 degrees). Further studies of the case will not be reported at this time.

Of the studies made, the following are selected for presentation here: in chart 2 *A*, blind spot for 0.5 degree blue, Dec. 21, 1929; in chart 2 *B*, blind spot for 0.5 degree blue, Jan. 7, 1930; in chart 2 *C*, *D* and *E*, blind spot for 1 degree blue and field for 0.5 degree blue mapped on the tangent screen, Jan. 24, Jan. 27 and May 23, 1930; in chart 2 *F*, blind spot for 0.17 degree white, May 23, May 27 and May 30; and in chart 2 *G*, blind spot for 0.5 degree blue, May 30.

CASE 2 (chart 3).—Mrs. K. G., aged 55, came under observation on Feb. 6, 1930, complaining of headaches in the temporal and occipital regions, vision slightly blurred at times, and redness and inflammation of the right eye. Ophthalmoscopic examination of the right eye showed many vitreous opacities, slight cupping and pallor of the disk at the lower temporal side; the rest of the nerve was of good color. The left eye showed slight general cupping of the disk, good color and slightly blurred margin. Tension in both eyes was 20; dilation of the pupil

produced no increase of tension. Vision in the right eye was 20/15 with +0.50 sphere —2.50 cylinder, axis 95 degrees; in the left eye vision was 20/15 with —1.12 cylinder, axis 75 degrees, with +2.25 sphere added for near vision in both eyes. Pilocarpine was prescribed on February 6. Under its action, subjective symptoms disappeared.

The examination with the tangent screen was made for the right eye on February 11 and for the left eye on February 12. The results for the left eye are shown in chart 3 *A*. The stimuli used were 0.5 degree white on black and 0.17 degree white on black. The former of these stimuli gave a blind spot of normal size and a scotoma extending from near the upper nasal rim of the blind spot in an irregular arc almost to the level of the horizontal meridian on the opposite side of the fovea. With this stimulus the blind spot and scotoma were not

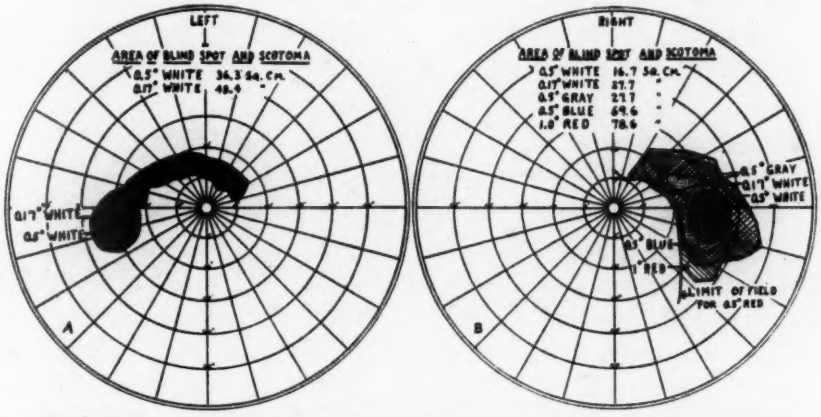

Chart 3 (case 2).—The blind spot and scotomatous extension when mapped with stimuli differing in diagnostic sensitivity. Left eye: The scotoma is detached from the blind spot for 0.5 degree white on black and continuous with the blind spot with 0.17 degree, white on black. Right eye: Great difference is seen in enlargement of the blind spot and size of the scotoma for form and color stimuli.

connected. The 0.17 degree stimulus gave a larger blind spot and scotoma than was obtained with the 0.5 degree stimulus. With the more sensitive stimulus, the scotoma was shown to be connected with the blind spot. The area of blind spot and scotoma, as projected on the tangent screen, is given for each stimulus in the chart.

The results for the right eye are given in chart 3 *B*. For this eye the stimuli employed were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 0.5 degree blue, 0.5 degree red and 1 degree red. For the 0.5 degree white stimulus, the blind spot was of normal size and shape; for the other stimuli it was greatly enlarged, the enlargement showing a pronounced tendency toward

an annular extension around the fovea. An inspection of the areas given in the chart shows the enlargement from greatest to least for the stimuli employed to be 1 degree red, 0.5 degree blue, 0.5 degree gray and 0.17 degree white, the two latter stimuli giving the same result. The blind spot could not be mapped with the 0.5 degree red stimulus because the limit of the field for this stimulus fell to the nasal side of the blind spot. The area of the blind spot and scotoma for 1 degree red, it will

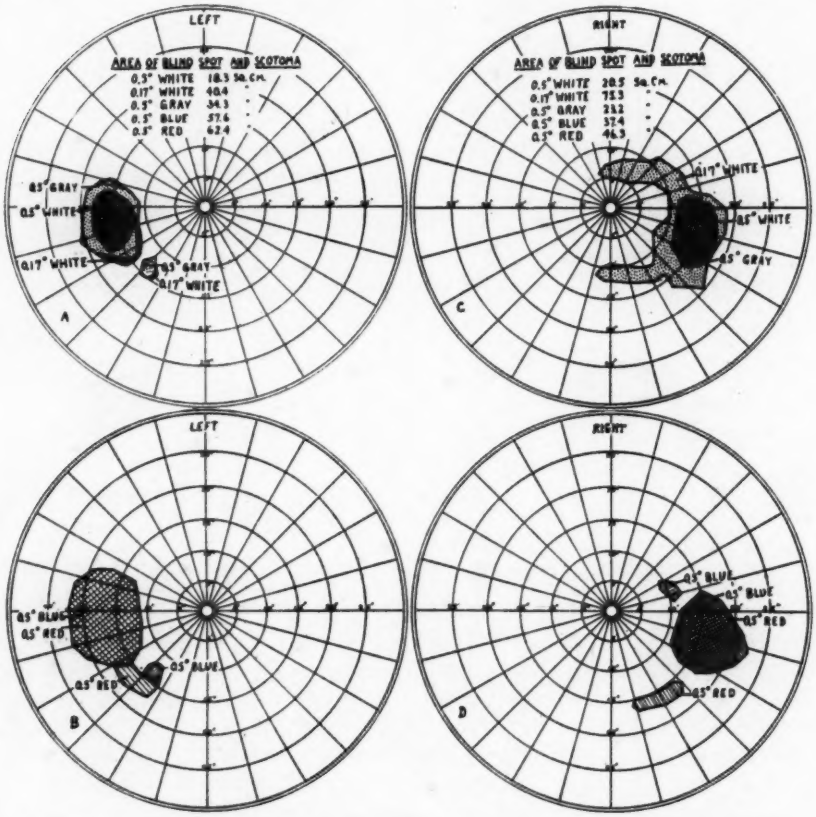

Chart 4 (case 3).—Glaucoma simplex in the right eye, and glaucoma simplex complicated with uveitis and posterior synechiae in the left eye. The blind spot and scotoma for stimuli differing in diagnostic sensitivity. There is a pronounced showing for 0.17 degree white on black and for the colored stimuli. The scotomas are detached for some stimuli; for others, they are continuous with blind spot.

be noted, is nearly five times as great as for the 0.5 degree white stimulus on black and nearly three times as great as for the 0.5 degree gray on black and 0.17 degree white on black.

CASE 3 (chart 4).—Mrs. B. N., aged 56, had glaucoma simplex in the right eye, and glaucoma simplex complicated with uveitis, and posterior synechiae in the left eye. In November, 1927, tension in the right eye was 44, and in the left eye 28. A miotic was prescribed. In spite of continuous use of miotics, the tension has

frequently measured between 30 and 35 in each eye. During 1928, the tension was higher in the right eye; during 1929, in the left eye. Posterior synechiae were noted in the left eye in January, 1930.

On March 8, 1930, ophthalmoscopic examination of the right eye showed clear media, glaucomatous cupping of the papilla, with good color of the papilla; the left eye showed an irregular pupil, synechiae above and nasally below, clumps of pigment granules in the anterior capsule, glaucomatous cupping of the papilla, and good color of the papilla.

Vision in the right eye was 20/20 + 3, with —0.25 sphere, —1.00 cylinder, axis 105 degrees; in the left eye, 20/15, with —0.25 sphere, —1.00 cylinder, axis 75 degrees.

An examination was made with the tangent screen on March 8, 1930. The stimuli employed were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 0.5 degree red and 0.5 degree blue. In the right eye (chart 4, *C* and *D*), there was no enlargement of the blind spot for 0.5 degree white on black or 0.5 degree gray on black. For 0.17 degree white on black, however, the blind spot was greatly enlarged with scotomatous extensions above and below the fovea extending beyond the middle of the field. The blind spot was enlarged also for 0.5 degree red and 0.5 degree blue, but not so much as for 0.17 degree white on black. This is the only instance in the cases studied thus far in which greater diagnostic sensitivity was shown for a form stimulus than for the color stimuli. It will be noted, however, that the color stimuli were not as small as the form stimulus in question. Detached scotomas were found for the color stimuli, above and nasalward for the blue and below and nasalward for the red. The scotoma for red was the larger. The contention is often made that the Bjerrum scotoma develops from the blind spot outward. Many cases are found in our work with a sensitized procedure in which the Bjerrum scotoma is shown to begin as a separate scotoma and later to merge with the blind spot. In the left eye (chart 4, *A* and *B*), there was no enlargement of the blind spot with a 0.5 degree white stimulus on black. Enlargement occurred, however, for both 0.5 degree gray (19 per cent) on black and 0.17 degree white on black; also small detached scotomas below and nasalward. Both the blind spot and the scotomas were slightly larger for the 0.17 degree white on black than for the 0.5 degree gray on black. For the 0.5 degree blue and 0.5 degree red stimuli, the enlargement of the blind spot was considerably greater than for the form stimuli, and scotomas occurred below and nasalward. The scotoma for the blue stimulus was detached from the blind spot; for the red stimulus, it had enlarged until it connected with the blind spot. For both right and left eye in this case a more extensive defect was shown for the red than for the blue stimulus.

CASE 4 (chart 5).—Mr. M. T., aged 40, had bilateral subacute glaucoma. There was also leukoma of the cornea of the right eye. Cyclodialysis was performed in April, 1929, following an acute attack of glaucoma with tension of 50 in both eyes. Since operation, the tension has remained within normal limits. Both eyes showed shallow cupping of the disk. The nerve head was not pale. Vision in the right eye was 20/40 + 2 with —0.25 sphere, —1.50 cylinder, axis 180 degrees; in the left eye, 20/15 with +0.25 cylinder, axis 90 degrees.

The examination with the tangent screen was made on Jan. 16, 1930. The stimuli used for the right eye were 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 1 degree red, 0.5 degree red, 1 degree blue, 0.5 degree blue and 1 degree green; for the left eye 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 0.5 degree red and 0.5 degree blue. For the right eye (chart 5*B*), the size of the blind spot for both of the form stimuli fell within the normal range—no

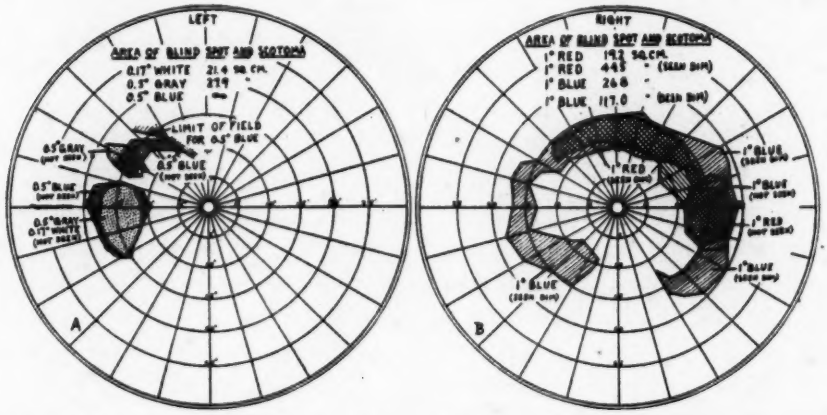

Chart 5 (case 4).—Bilateral subacute glaucoma and leukoma of the cornea of the right eye. The blind spot and scotoma for stimuli differing in diagnostic sensitivity. Left eye: There is a detached scotoma for 0.5 degree gray (19 per cent) on black and 0.5 degree blue; the scotoma for 0.5 degree blue merges with blind area due to contracting field. Right eye: Pronounced arcuate scotoma for 1 degree red and 1 degree blue, continuous with blind spot; the blind spot is normal and there are no scotomas for form stimuli.

scotomatous extensions. For 1 degree red, there was a large scotomatous extension above running nearly halfway around the field. For 1 degree blue, the scotomatous extension was both above and below and nearly encircled the fovea. In both of these scotomas for color, the stimulus was seen dim. The blind spot could not be mapped with the 0.5 degree red, 0.5 degree blue, or 1 degree green stimuli because the involvement had caused so much contraction of the fields that the limits fell to the nasal side of the blind spot. In such cases as this the presupposition is that the contraction of the field is due both to the enlarge-

ment of the scotoma and to a progressive loss of sensitivity from the periphery toward the center. In the left eye (chart 5*A*), the blind spot for both form stimuli used was normal as to size and shape. For 0.5 degree gray, however, there was a detached scotoma of considerable size above and to the nasal side of the blind spot. There was a slight indication in this region of insensitivity to the 0.17 degree white stimulus also; but the area of the insensitivity was too indefinite to be mapped. A detached scotoma for 0.5 degree blue, however, was found in this region which extended farther toward the nasal side of the field and merged with the blind area produced by the contraction of the field. No scotomatous involvement was found for 0.5 degree red.

CASE 5 (chart 6).—Mrs. B. S., aged 48, had bilateral subacute congestive glaucoma. She came under observation on June 29, 1928, complaining of attacks of pain. Tension was elevated in the right eye and normal in the left. A corneoscleral trephine was performed in the right eye on July 6, 1928. While in the hospital, a slight increase of tension occurred in the left eye, which subsided promptly under the administration of pilocarpine. There was a history of occasional attacks of pain and increased tension in the left eye until February, 1929, when a trephine with a graft in Tenon's capsule was performed. On Jan. 27, 1930, the tension was normal, the eyes clear, and there was good drainage in each eye. The fundi were normal. Vision was 20/30 in both eyes with +1.25 sphere, +0.50 cylinder, axis 180 degrees, with +1.75 spheres added for near vision.

The examination with the tangent screen was made on Jan. 24, 1930. The stimuli employed were, for both eyes, 0.5 degree white on black, 0.17 degree white on black, 1 degree each red, green and blue, and 0.5 degree red and blue. In the right eye (chart 6, *E*, *F*, *G* and *H*), the blind spot was normal for 0.5 degree white. It was more than twice as large for 0.17 degree white, about three times as large for 1 degree blue and more than three times as large for 1 degree green. The blind spot for 1 degree red was much smaller than for blue and green and fell within the normal range of sizes. For 1 degree blue, a detached scotoma was found below and to the nasal side of the blind spot. For the 0.5 degree red and blue, the limits of the field fell to the nasal side of the blind spot. In the left eye (chart 6, *A*, *B*, *C* and *D*), the blind spot was normal for the 0.5 degree white stimulus and enlarged for 1 degree green, 1 degree blue, 1 degree red and 0.17 degree white. For the 1 degree blue, there was a scotomatous extension down and to the nasal side. This extension was continued for some distance as a faint scotoma, i.e., in this region the stimulus was seen dim. For 0.5 degree blue, the limit of the field fell to the nasal side of the blind spot. For 0.5 degree red, there was a pronounced enlargement of the blind spot, with scotomatous extensions above and below. In these extensions, the stimulus

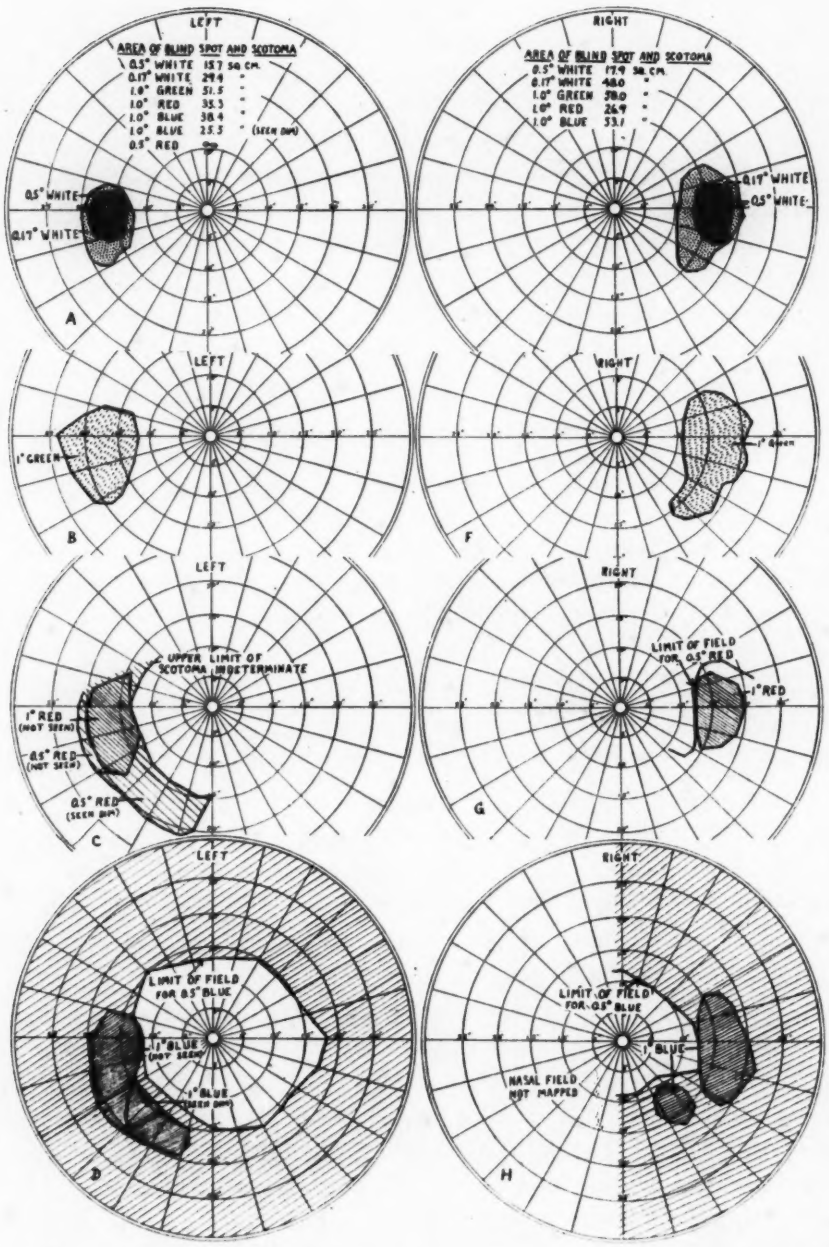

Chart 6 (case 5).—Bilateral subacute congestive glaucoma. A striking demonstration of the superior diagnostic sensitivity of color as compared with form stimuli.

was seen dim. The lower one extended down and around the fovea to approximately the middle of the field; the upper was indefinite in extent.

CASE 6 (chart 7).—Miss F. N., aged 64, had glaucoma simplex. Tension under treatment with pilocarpine has been slightly above normal for the past two years. Blurring of vision occurred practically daily in the right eye. There was slight general cupping of the disk involving the whole nerve head. The temporal side was slightly pale. There was a thin halo around the margin of the disk. In the left eye the superior nasal edge of the disk was blurred. There was slight cupping, limited to a small portion on the temporal side. No halo was seen. Vision in the right eye was 20/15-3 with +1.00 sphere, +0.50 cylinder, axis 180 degrees; in the left eye, 20/15-3 with +2.50 sphere, +0.75 cylinder, axis 180 degrees, with 2.25 sphere added for near vision in both eyes.

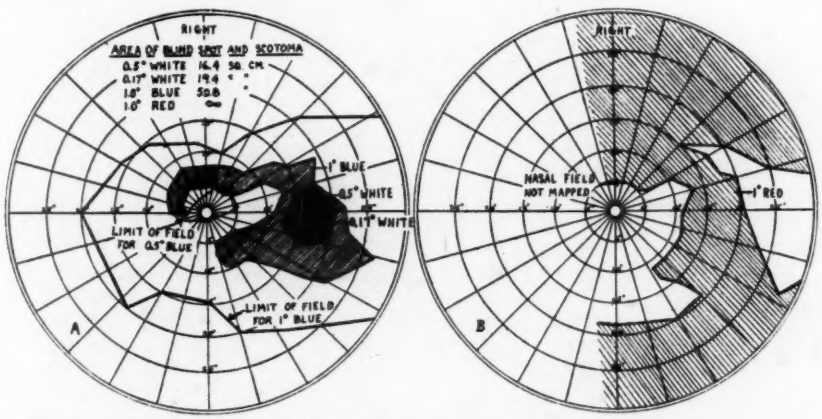

Chart 7 (case 6).—Glaucoma simplex. The blind spot is normal for 0.5 and 0.17 degrees white on black. There is a large arcuate scotoma above and below for 1 degree blue; the field is contracted within the 5 degree isopter for 0.5 degree blue. For 1 degree red, scotomatous extension of blind spot merges above and below with blind area due to contraction of field.

The study with the tangent screen was made on Jan. 31, 1930. Results for only the right eye are given here. The stimuli used were 0.5 degree white on black, 0.17 degree white on black, 1 degree blue, 1 degree red and 0.5 degree blue. For 0.5 degree white and 0.17 degree white, the blind spot was not enlarged. For 1 degree blue, it was greatly enlarged with scotomatous extensions above running halfway around the fovea above and nearly one-fourth the way around the fovea below. For 1 degree red, there was still greater enlargement, the scotoma spreading above and below to cover the greater part of the field. Sensitive areas were left, however, on both the temporal and the nasal side of the blind spot, the area on the nasal side including the fovea. For 0.5 degree blue, the field was contracted to within 5 degrees of the center.

CASE 7 (chart 8).—Miss D. K., a Negro, had bilateral glaucoma simplex. She came under observation on Feb. 7, 1930. Ten days earlier, she noticed inflammation of the eye and dimness of vision. Both disks were sharply cupped. The vessels were pushed somewhat to the nasal side, especially in the left eye. Tension was around the upper limit of normal in both eyes. Pilocarpine was prescribed. Vision in the right eye was 20/20 with —0.25 cylinder, axis 120 degrees; in the left eye, 20/20 with —0.25 sphere, with +2.25 sphere added for near vision in both eyes.

The examination with the tangent screen was made on Feb. 28, 1930. The stimuli used were for the right eye, 0.5 degree white on black,

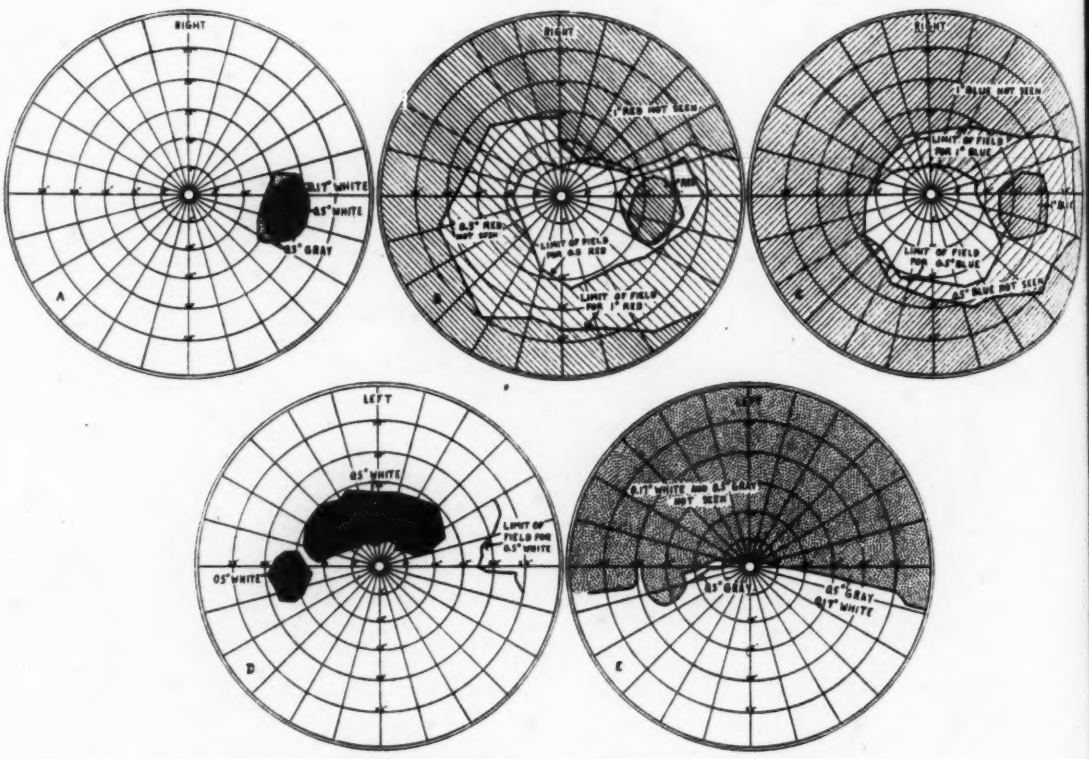

Chart 8 (case 7).—Bilateral glaucoma simplex. Right eye: The blind spot is normal for form stimuli; extensive involvement and loss of sensitivity for color stimuli. Left eye: There is a large detached scotoma for 0.5 degree white; loss of the entire upper half of the field for 0.17 degree white and 0.5 degree gray on black. The examination was not made for color.

0.5 degree gray (19 per cent) on black, 0.17 degree white on black, 1 degree red, 1 degree blue, 0.5 degree red and 0.5 degree blue; for the left eye, 0.5 degree white on black, 0.5 degree gray (19 per cent) on black and 0.17 degree white on black. In the right eye (chart 8, *A*, *B* and *C*), the blind spot showed no enlargement for 0.5 degree white on black, 0.5 degree gray on black and 0.17 degree white on black. For

red, a large scotomatous area seems to have formed above the blind spot, merging peripherally with the blind area produced by the contracting field. For the 1 degree stimulus, this scotoma was completely detached from the blind spot; for the 0.5 degree stimulus, it merged with the blind spot. For both stimuli, the field for red showed irregular contraction. The field for blue also showed irregular contraction. For the 0.5 degree stimulus, its temporal limits fell well inside the nasal limit of the blind spot. Between the limits of the field for the 1 degree and the 0.5 degree blue stimulus was an area insensitive to 0.5 degree blue that gave the appearance of an enlargement of the blind spot into a scotomatous extension which passed nearly around the fovea above and below. In the left eye (chart 8, *D* and *E*), with 0.5 degree white on black, a large scotoma was found above and to the nasal side of the blind spot. With 0.5 degree gray and 0.17 degree white on black,

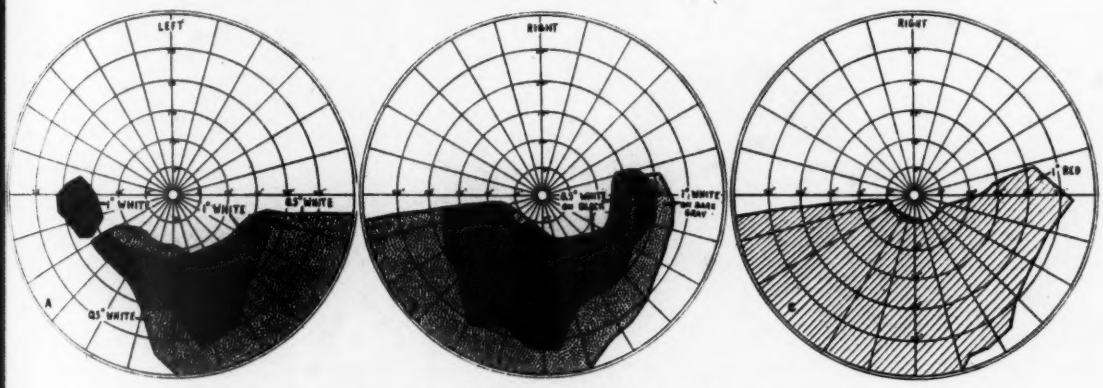

Chart 9 (case 8).—Bilateral chronic glaucoma simplex. Left eye: There is a large detached scotoma for 1 degree white on black and much larger for 0.5 degree white on black. Right eye: for 0.5 degree white on black there is a large arcuate scotoma below, which was more than doubled in size for 1 degree white on dark gray (10 per cent); for 1 degree red, scotomatous extension of blind spot covers nearly all of the lower half of the field.

this scotoma was merged with the blind spot and enlarged to include more than the entire upper half of the field. With 0.5 degree white on black, a nasal step of about 6 degrees was found.

CASE 8 (chart 9).—Mr. G. N. had bilateral chronic glaucoma simplex. There was a history of tension ranging between 40 and 46 in both eyes. At the time the examination was made with the tangent screen, the tension had been reduced to about 20. The nerve head was deeply cupped in both eyes. The vessels were crowded to the nasal side of the disk. Vision in the right eye was 20/20-3 with +0.75 sphere, +0.25 cylinder, axis 180 degrees; in the left eye, 20/15-3 with +1.00 sphere, with +2.75 sphere added for near vision in both eyes.

The examination with the tangent screen was made on Feb. 24, 1930. The stimuli used were for the right eye 0.5 degree white on black, 1

degree white on dark gray (coefficient of reflection 10 per cent) and 1 degree red; for the left eye, 1 degree white on black and 0.5 degree white on black. In the right eye (chart 8, *B* and *C*), the blind spot for 0.5 degree white on black was enlarged below into a broad scotomatous extension which passed nearly halfway around the field. The scotomatous extension for 1 degree white on dark gray took a similar course but was much larger. For 1 degree red, the enlargement of blind spot and scotoma was greater still and extended over a part of its course nearer than 5 degrees from the fovea. In the left eye (chart 8 *A*), with 1 degree white on black, a large scotoma was found below and to the nasal side of the blind spot. With 0.5 degree white on black, this scotoma was more than doubled in size. The scotoma for both stimuli

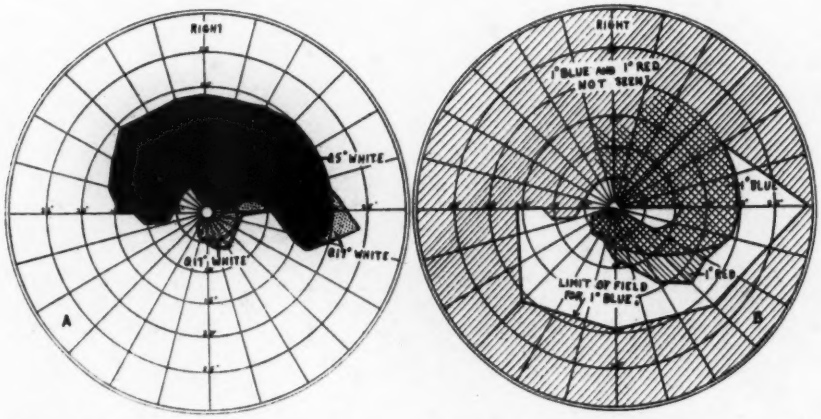

Chart 10 (case 9).—Chronic glaucoma simplex in the right eye. The blind spot is enlarged into a broad arcuate scotoma above for 0.5 degree white on black. For 0.17 degree white on black, this scotoma is somewhat larger with a narrow extension nearly encircling the fovea below. For 1 degree blue and 1 degree red, the scotoma merges above with the blind area due to the contracting field; the upper half of the field is almost entirely gone; sensitivity in the lower half is limited to the narrow arcuate strip. Extension below encroaches on the fovea and nearly encircles it.

were detached from the blind spot. The area separating the scotoma from the blind spot was found to be sensitive also to 0.17 degree white on black.

Case 9 (chart 10).—Mr. T. F., aged 41, had chronic glaucoma simplex in the right eye, and secondary cataract in the left eye. He came under observation in December, 1925. The vision was completely gone in the left eye. There was a history of failing vision in the right eye for one year. From 1925 to 1929, he had had several operations which did not succeed in reducing tension to normal limits. On Jan. 2, 1930, the tension was 30. The disk was cupped and pale. The vision was approximately 7/40.

The examination of the right eye was made with the tangent screen on Jan. 8, 1930. The stimuli used were 0.5 degree white on black, 0.17 degree white on black, 1 degree red and 1 degree blue. For 0.5 degree white, the blind spot was enlarged into an extensive scotoma above extending more than halfway around the field and from near the fovea to an excentricity of 20 degrees. For 0.17 degree white, this scotoma was shown to extend also almost around the fovea below and to approach even more closely to the fovea from below than from above. For 1 degree red and 1 degree blue, the involvement was so great that almost the entire field was blind, and for both the encroachment in the central field was still greater than for the form stimuli. The area blind to red was somewhat greater than the area blind to blue.

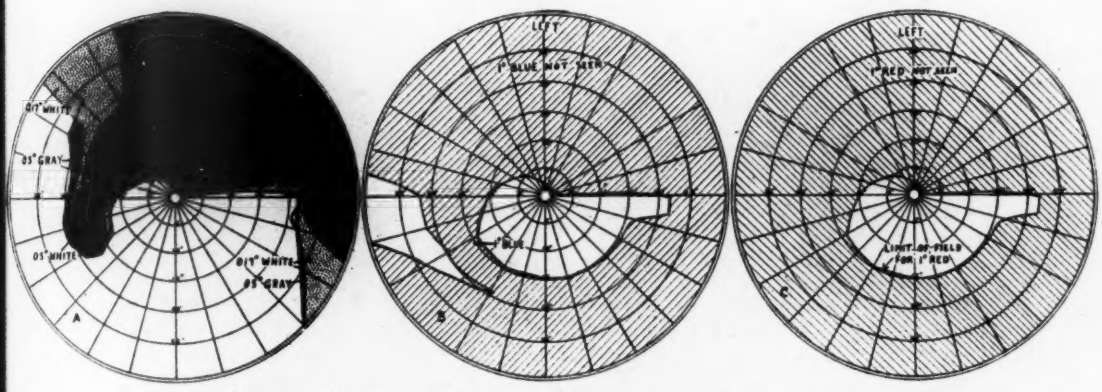

Chart 11 (case 10).—Bilateral chronic glaucoma simplex. For 0.5 degree white on black, there is an enlarged blind spot with a large fan-shaped scotomatous extension above. For 0.17 degree white on black, there is a much larger scotoma. The scotoma is slightly larger still for 0.5 degree gray (19 per cent) on black. For 1 degree blue and 1 degree red, the scotoma is enlarged to include nearly all the upper field and the greater part of the lower field. For all stimuli, there is a large nasal step.

CASE 10 (chart 11).—Mrs. F. K., aged 60, had bilateral chronic glaucoma simplex. Corneoscleral trephine in the left eye was done on Jan. 27, 1929. Details of the fundus could not be seen owing to diffuse nuclear opacities of the lens. Vision in the right eye was 20/30-1 with +0.50 cylinder, axis 45 degrees; in the left eye, 20/30-1 with —1.00 sphere, —1.50 cylinder, axis 95 degrees.

An examination of the left eye with the tangent screen was made on Jan. 22, 1930.[6] The stimuli used were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 1 degree red and 1 degree blue. The case was too advanced to permit of the use of smaller colored stimuli. For 0.5 degree white, the blind

6. The examination of this case was made by Dr. Marion R. Stoll of this laboratory.

spot showed an enormous scotomatous extension upward to the nasal side of the field, merging with the blind area produced by the contraction of the field and encroaching closely on the fovea. The limits of the sensitive area took the form of a deep nasal step. There was also the beginning of a scotomatous extension below the blind spot. For 0.17 degree white, there was a marked enlargement of this scotoma and a still greater enlargement for 0.5 degree gray on black. For 1 degree red and 1 degree blue, the whole upper half of the field was gone, with the exception of a small sensitive patch around the fovea, and the scotomatous extension below passed clear around the fovea merging in all directions with the blind area produced by the contracting field. The sensitivity to red in the lower half of the field was confined to an area lying almost entirely within 15 degrees of the fovea. For blue, in addi-

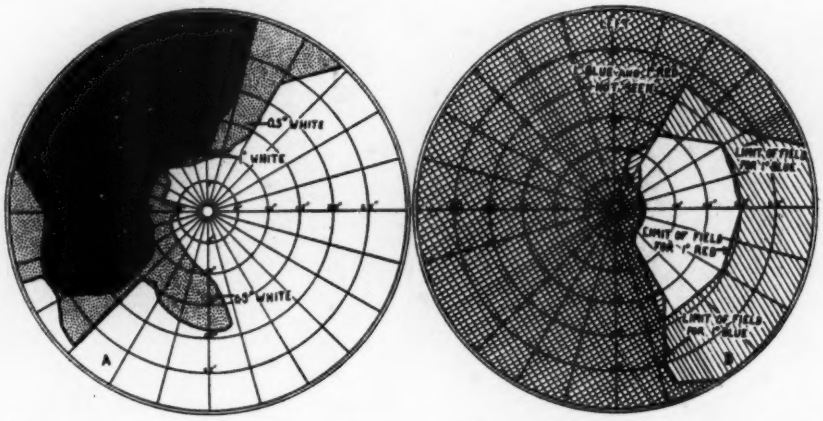

Chart 12 (case 11).—Healed perforatory wound of the ciliary body of the right eye; sympathetic ophthalmia of the left eye. For 1 degree white, the blind spot is greatly enlarged with a broad scotomatous extension above. The scotoma is much larger for 0.5 degree white on black; there is also a broad arcuate extension below. For 1 degree blue, there is only an irregular patch of sensitivity left in the nasal half of the field; there is a much smaller patch for 1 degree red. The fovea is involved for both color stimuli.

tion there was a small sensitive area in the temporal half of the field lying almost entirely below the horizontal meridian and beyond 20 degrees from the center. For the colored stimuli, there was also a pronounced nasal step.

CASE 11 (chart 12).—Mr. M. N., aged 30, had healed perforatory wounds of the ciliary body in the right eye, and sympathetic ophthalmia of the left eye. On Jan. 3, 1930, six weeks after injury to the right eye, the left eye became painful and inflamed. Ophthalmoscopic examination of the right eye revealed apparently clear media; a rather hazy fundus, edematous and slightly foggy in appearance; a much blurred disk margin, which could not be seen clearly. There were no

exudates. The vessels were slightly attenuated, blurred in places but distinct farther along. The left eye revealed apparently clear media, although the vitreous was hazy. The nerve head was entirely obscured, except for the place of exit of the vessels, and was definitely elevated. There were patches of choroiditis below the disk. The vessels were rather indistinct, owing to edema of the retina. On January 3, vision in the right eye was 20/70 with +1.00 sphere; in the left, 20/70 with +1.50 sphere. On February 7, vision in the right eye was 4/70; in the left, 20/100.

An examination of the left eye with the tangent screen was made on Jan. 21, 1930. The stimuli used were 1 degree white on black, 0.5 degree white on black, 1 degree red and 1 degree blue. For 1 degree white, the blind spot expanded into a large scotoma which extended into the upper half of the field broadening out temporalward and nasalward and merging with the blind area produced by the contraction of

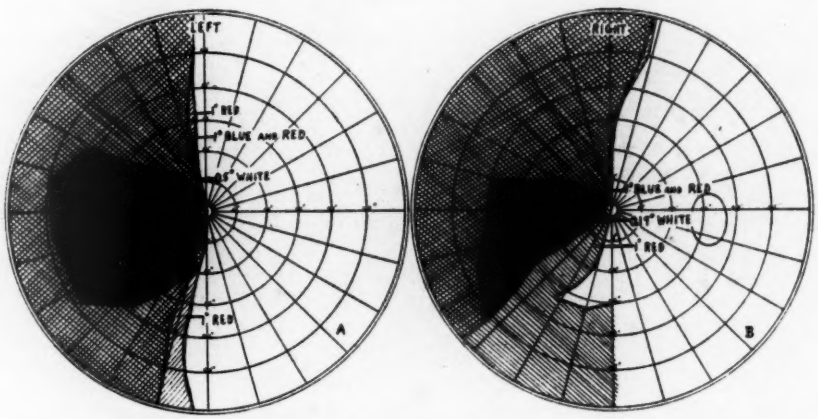

Chart 13 (case 12).—Syphilitic vascular lesion probably of the suprageniculate optic nerve pathway on the right side. There is a large homonymous scotoma for form stimuli, not nearly so large in the right eye for 0.17 degree white on black as in the left eye with 0.5 degree white on black. Homonymous hemianopia is seen for 1 degree red, the involvement being slightly less in the right eye. Involvement for 1 degree blue is not so great in either eye, but much greater in the left than in the right eye.

the field. For 0.5 degree white, the extension was continued temporalward and nasalward above, and in addition, downward and nasalward with a closer approach to the fovea. For 1 degree blue, the whole of the temporal and a large part of the nasal field were involved, including the fovea. For red, the destructive process had continued until for the 1 degree stimulus only a comparatively small area of sensitivity was left, extending from 5 degrees to 20 degrees on the nasal side of the fovea.

Case 12 (chart 13).—Mr. A. D., had a syphilitic vascular lesion probably of suprageniculate optic nerve pathway on the right side. He came under observation

on Jan. 27, 1930. Ophthalmoscopic examination of the right eye showed the superior and nasal margin of the nerve slightly blurred; there was slight thinning of the retina and choroid on the temperal side, and one small spot of cholesterol on the temporal margin of the disk; the remainder of the fundus was clear. There was marked pulsation of the superior temporal vein, but no venous compression; the vessel walls were a trifle pronounced; there was no sign of decided retinal angiosclerosis. A similar report was made on the left eye. There was weakness of convergence. The pupils acted sluggishly in both direct and consensual reaction. There was a strongly positive Wassermann reaction of the spinal fluid. The patient had a long history of unsuccessful diagnosis. It was chiefly because of the results of the visual field studies that a Wassermann test of the spinal fluid was ordered. Vision in the right eye was 20/15 with —0.50 sphere, —0.50 cylinder, axis 165 degrees; in the left eye, 20/20-1 with —1.00 sphere, —0.25 cylinder, axis 180 degrees.

The examination with the tangent screen was made on Jan. 28, 1930. The stimuli used were 0.5 degree white on black, 0.17 degree white on black, 1 degree red and 1 degree blue. A left homonymous paracentral scotoma was found for white and a left homonymous hemianopia for red and for blue. In the left eye (chart 13 *A*), the scotoma which merged with the blind spot was mapped most successfully with 0.5 degree white. For 0.17 degree white it was somewhat larger but had less well defined limits. In the right eye (chart 13 *B*), the scotoma was smaller and less dense and was mapped more successfully with 0.17 degree white than with 0.5 degree white. Both sizes of stimulus, however, were occasionally seen in minute patches within the scotoma of each eye. The fovea of neither eye was involved in the scotoma, but the defect in the left eye approached more nearly to the fovea than in the right. For both 1 degree red and 1 degree blue, left hemianopias were found, the left half of the fovea being involved in the defect for red in both eyes and in the defect for blue in the right eye. The scotoma for color was larger in the right than in the left eye and more extensive in both eyes for red than for blue.

Case 13 (chart 14).—Miss N. H., aged 32, had axial neuritis. She came under observation on March 23, 1930. Several weeks prior to this time, failure of vision with some pain in the left eye was noticed. Ophthalmoscopic examination showed the fundi to be normal in appearance. There was a history of amenorrhea for the past nine years. Vision in the right eye was 20/20-1 with +2.00 sphere, +0.25 cylinder, axis 90 degrees; in the left eye, 20/100 with +1.00 sphere, +.50 cylinder, axis 90 degrees. We found marked contraction in the temporal and upper temporal fields with 1 degree white on black and 1 degree gray (19 per cent) on black. The contraction was greater with the gray than with the white stimulus.

There was complete bitemporal hemianopia with 1 degree red; temporal hemianopia in the right eye and loss of the upper temporal field in the left eye with 1 degree blue. Studies with the tangent screen also showed pronounced functional defects in the temporal field of both eyes, more pronounced in the right than in the left eye. On the basis of results with perimeter and tangent screen and the history of amenorrhea, a roentgen examination was ordered. This examination showed destruction of the sella turcica.

Only the examination of the left eye with the tangent screen will be reported here. The stimuli used were 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 1 degree red, 0.5 degree red, 1 degree blue and 0.5 degree blue. There was considerable enlargement of the blind spot for 0.17 degree white. For 0.5 degree gray, the blind spot merged with a large sector-shaped scotoma the apex of which extended into the fovea. Over a part of this area, indicated on the chart,

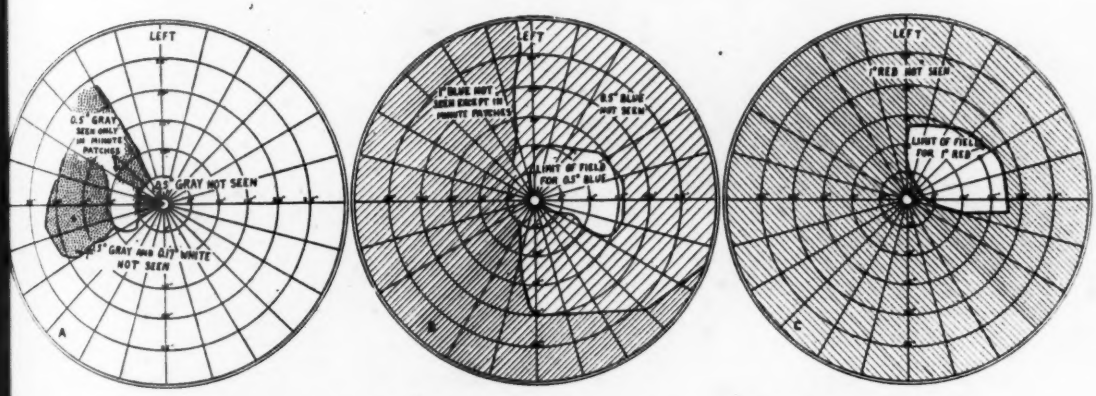

Chart 14 (case 13).—Axial neuritis. There is an enlarged blind spot and temporal scotoma for 0.17 degree white on black; a much larger scotoma encroaching on the fovea for 0.5 degree gray (19 per cent) on black. There is loss of the temporal field beyond the 5 degree isopter for 1 degree blue; also loss of the temporal and nearly all of the nasal field including the fovea for 0.5 degree blue and 1 degree red.

the stimulus was seen here and there in minute patches. In the entire upper temporal half of the field beyond 5 degrees, 1 degree blue was seen also only here and there in patches. For 0.5 degree blue there remained only a small patch of sensitivity above and to the nasal side of the fovea. The fovea was blind to this size of blue stimulus. For 0.5 degree red, sensitivity was gone over the entire field. For 1 degree red, there remained a small patch of sensitivity above and to the nasal side of the fovea.

Case 14 (charts 15 and 16).—Mr. K. Y., aged 28, a Negro, had bilateral optic neuritis. He came under observation on April 24, 1930. About a week prior to this, he began to have severe frontal pain over the eyes, which persisted

for three days before dimming of vision was noticed. The pain became less severe, but vision gradually decreased. Ophthalmoscopic examination of the right eye revealed: a red reflex; clear media; blurred nasal margin of the disk; slight cloudiness with grayish exudate, which extended a short distance over the retina; some atrophy on the temporal side of the disk; perivasculitis of the vessels about the disk; edema of the retina adjacent to the disk on the nasal side. Markings of the lamina cribrosa and vascular funnel were not seen. There was a temporal pigment crescent. The veins were distended and tortuous near the disk. The macula was not abnormal in appearance. No hemorrhages were seen. On exami-

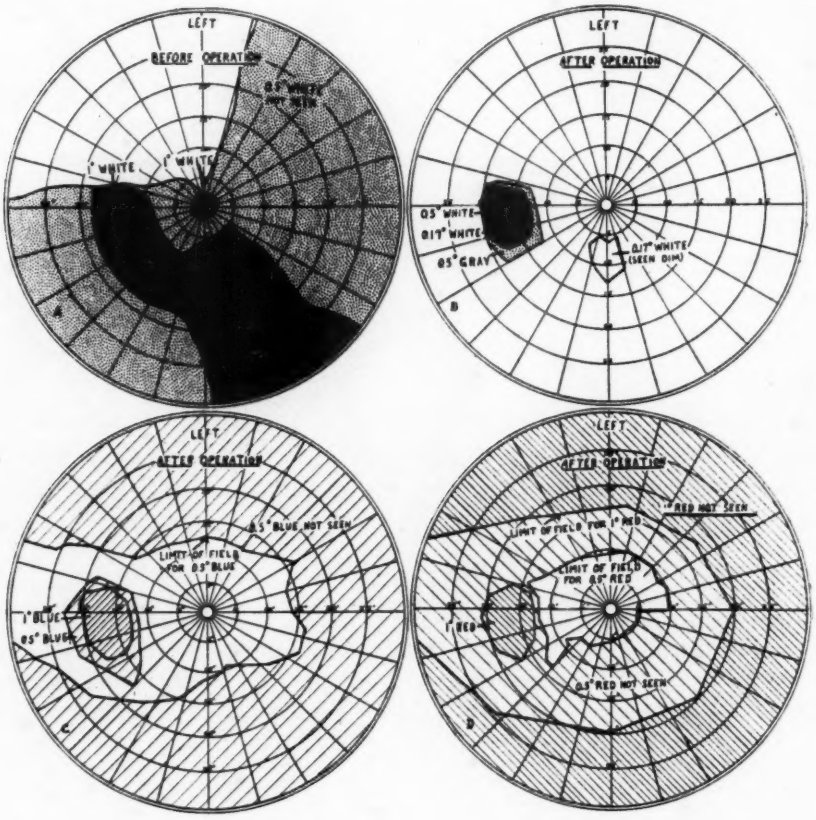

Chart 15 (case 14).—Bilateral optic neuritis. Left eye: Before operation no color was seen with stimuli of ordinary visibility. For 0.17 degree white on black, the blind spot is greatly enlarged, expanding into the broad scotoma below. There is a small central scotoma. For 0.5 degree white on black the lower half of the field is blind and nearly all of the upper nasal quadrant. After operation: For 0.5 degree and 0.17 degree white on black, the blind spot is normal; for 0.5 degree gray (19 per cent) on black it is somewhat enlarged. There is a small patch below the center of the field in which 0.17 degree white on black is seen dim. The blind spot is enlarged for 0.5 degree blue, but not enlarged for 1 degree blue and 1 degree red. A large part of the field is blind to 0.5 degree blue and 1 degree red. Sensitivity is left only in the small central area for 0.5 degree red.

nation of the left eye, the fundus appeared the same as in the right eye, with the same degree of involvement. On April 24 vision in the right eye, without correction, was 20/70; in the left eye, 10/200; on April 28, in the right eye, 8/200 with 0.50 cylinder, axis 75 degrees; in the left eye, 3/200 with 0.50 cylinder, axis 105 degrees. The vision decreased noticeably during the following days. The Wassermann reaction of the spinal fluid was negative. A stereoroentgenogram of the skull showed the sella turcica to be normal; the ethmoids, sphenoids and frontal bone were apparently normal. It was decided, however, to open the sinuses. On May 6, the posterior half of each turbinate was removed, and the posterior ethmoids and sphenoids were opened. No free pus, but some thickened membrane, was found; there was more of this on the right than on the left side. Material from the right sphenoid was tested. Culture yielded a light growth of *Staphylococcus aureus.*

On May 16, ophthalmoscopic examination of the right eye showed a red reflex; clear media; slightly blurred nasal margin of the disk; slight perivasculitis of the

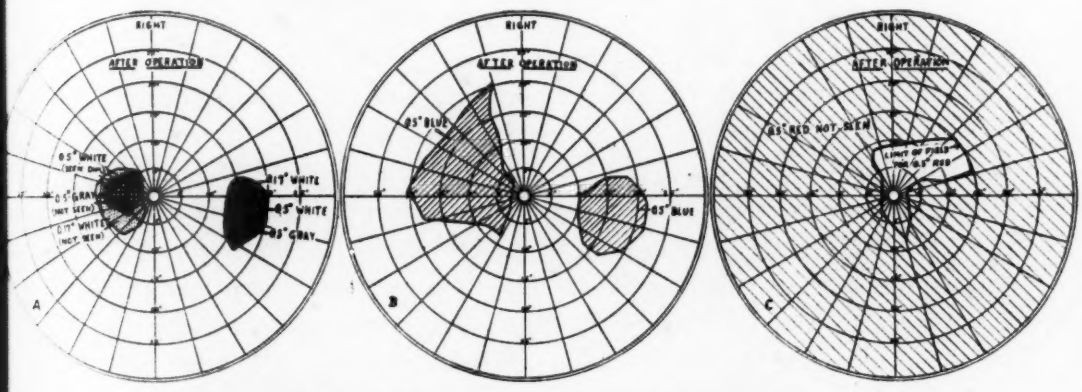

Chart 16 (case 14).—Right eye: Before operation, only finger movements were seen. After operation: The blind spot is normal for all the form stimuli; near the fovea on the nasal side there is a scotoma for 0.5 degree gray (19 per cent) on black; in part of this 0.5 degree white is seen dim. The scotoma is enlarged for 0.17 degree white; there is pronounced enlargement of the blind spot for 0.5 degree blue, also a large scotoma at the nasal side of the fovea. For 0.5 degree red only a small centrally located patch of sensitivity remains.

vessels emerging from the disk; a slight temporal pigment crescent and normal veins. The condition of the left eye was the same as that of the right, but less pronounced. Vision in the right eye was 20/40 with +0.50 cylinder, axis 75 degrees; in the left eye, 20/20 with +0.50 cylinder, axis 145 degrees.

Before the operation, an examination of the right eye could not be made with the tangent screen because of poor vision; only finger movements could be seen. In the left eye (chart 15), there was no color vision for stimuli of ordinary visibility. The patient reported that every-

thing, colored and colorless, appeared yellowish. An examination with the tangent screen was made on May 2, 1930. The stimuli used were 1 degree white on black and 0.5 degree white on black. For 1 degree white, there was a small central scotoma, irregular in shape, which extended around the fovea on the temporal side, including the blind spot, to a point 15 degrees above the horizontal meridian and obliquely downward toward the nasal side until it merged with the blind area produced by the contracting field. For the 0.5 degree white, all of the lower half of the field was blind and nearly all of the upper nasal quadrant.

Following the operation, an examination was made with the tangent screen on May 13 and 14. The stimuli used for the left eye were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 1 degree red, 1 degree blue, 0.5 degree red and 0.5 degree blue. In the left eye (chart 15), the blind spot was normal for 0.5 degree white and 0.17 degree white. For 0.5 degree gray, it was somewhat enlarged. There was also a scotomatous area of irregular shape below the center of the field, in which the 0.17 degree white stimulus was seen dim. For 1 degree blue, the blind spot showed little if any enlargement. For 0.5 degree blue, however, there was enlargement. There was also a pronounced contraction of the field for 0.5 degree blue, particularly in the upper, lower and nasal meridional quadrants. For 1 degree red, there was little if any enlargement of the blind spot, but there was again a marked contraction of the field, particularly in the vertical and nasal meridional quadrants. The blind spot could not be mapped with the 0.5 degree red because of the contraction of the field, which was great for this stimulus, the limits coming at places within 5 degrees of the center of the field. In the right eye (chart 16) there was no enlargement of the blind spot for any of the form stimuli, white or gray. Near the fovea on the nasal side was an absolute scotoma for 0.5 degree gray which was much larger for 0.17 degree white. Over the greater part of the area which was blind to 0.5 degree gray, 0.5 degree white was seen dim. There was a pronounced enlargement of the blind spot for 0.5 degree blue. Also, there was for this stimulus a large scotoma of irregular shape near the fovea on the nasal side. Almost the entire field was blind to 0.5 degree red. A small sensitive patch remained above and to the temporal side of the fovea, from the nasal end of which a small extension dipped down to about 7 degrees below the fovea.

CASE 15 (chart 17).—Mr. G. P., aged 56, had toxic axial neuritis. He came under observation on May 2, 1930, complaining of inability to read. The eyeballs ached at times. He had no headaches. His vision had been failing for about a month. Colors had not looked natural for about three months. Ophthalmoscopic examination showed a normal appearance of the media and fundus. There were no objective signs of abnormality. Vision in the right eye with a +1.50 sphere, +0.50 cylinder, axis 90 degrees was 20/30 + 1 on May 3 and 20/70 on May 21; in the left eye with a +1.50 sphere, +0.50 cylinder, axis 90 degrees, 20/30 on May 3 and May 21.

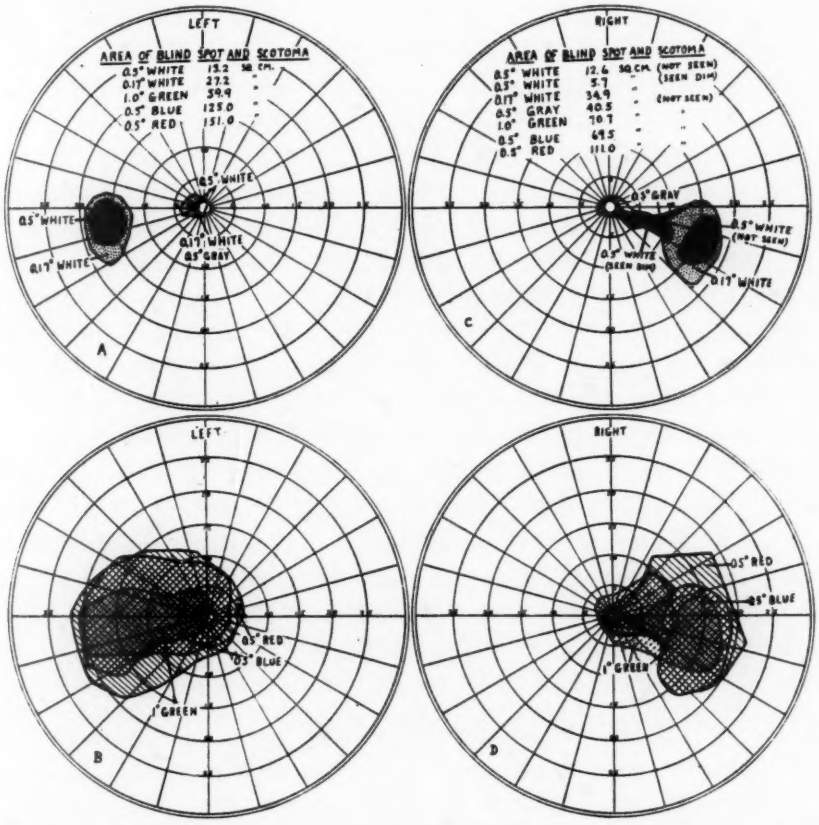

Chart 17 (case 15).—Toxic axial neuritis. Left eye: For 0.5 degree white on black, the blind spot is normal, with a small scotoma to the nasal side of the fovea. For 0.17 degree white on black, the blind spot is enlarged. The scotoma is enlarged and encroaches on the fovea for both 0.17 degree white and 0.5 degree gray on black. The blind spot and scotoma are greatly enlarged in order for 1 degree green, 0.5 degree blue and 0.5 degree red. Right eye: For 0.5 degree white on black, the blind spot is normal. For 0.17 degree white on black, the blind spot is greatly enlarged with a finger-shaped scotoma involving the fovea. There is slightly greater enlargement of the blind spot and scotoma for 0.5 degree gray on black. Over part of this scotoma, 0.5 degree white is seen dim. The blind spot and scotoma are greatly enlarged in order for 0.5 degree blue, 1 degree green and 0.5 degree red. Involvement for form is greater in this eye; for color, it is less.

The examination was made with the tangent screen on May 21. For both eyes the stimuli used were 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, 0.5 degree red, 0.5 degree blue and 1 degree green. In the right eye, the blind spot showed no enlargement for 0.5 degree white. For 0.17 degree white, however, there was considerable enlargement of the blind spot, with a finger-shaped scotomatous extension which involved the fovea. For 0.5 degree gray, the scotomatous extension was still larger, particularly about the fovea. Over a part of this scotoma from a point near the fovea to beyond 10 degrees, 0.5 degree white was seen dim. For 0.5 degree blue, the blind spot was greatly enlarged. There was also a narrow scotomatous extension involving the fovea. For 0.5 degree red, the enlargement of the blind spot was still greater and the scotomatous extension much broader. For 1 degree green, the enlargement of the blind spot was not nearly so great as for 0.5 degree red and 0.5 degree blue, but the scotomatous extension was much larger. In the left eye, there was some enlargement of the blind spot for 0.17 degree white but none for 0.5 degree white. In addition, there was a small scotoma for 0.5 degree white to the temporal side of the fovea. For 0.17 degree white and 0.5 degree gray, this scotoma was much larger and involved nearly all of the fovea. While the involvement for the form stimuli was much less in this eye than in the right eye, the involvement for the color stimuli was considerably greater. For 0.5 degree blue, the blind spot enlarged into a broadly oval scotoma which extended about 6 degrees beyond the fovea. For 0.5 degree red, this scotoma was not so broad and did not extend so far beyond the fovea. For 1 degree green, the scotoma was considerably narrower and extended only to the center of the field.

A brief analysis of the foregoing results shows the following facts. Of the thirteen eyes examined in an incipient stage with 0.5 degree white on black and with the colored stimuli, only one would have shown a defect if the examination had been made with the form stimulus alone; all showed a pronounced defect when examined with the colored stimuli. Of the ten eyes examined in an incipient stage with 0.5 degree gray (19 per cent) on black and with the colored stimuli, only six would have shown a defect if examined with the form stimulus alone; all showed a pronounced defect when examined with the colored stimuli. Of the fourteen eyes examined in an incipient stage with the 0.17 degree white on black and with the colored stimuli, only eight would have shown a defect if examined with the form stimulus alone; all showed a pronounced defect with the colored stimuli. Of the nine eyes examined

in an incipient stage with all three form stimuli, only one showed a defect with 0.5 degree white on black, five with 0.5 degree gray (19 per cent) on black, and six with 0.17 degree white on black. Of the twelve eyes examined in a more advanced stage with both form and colored stimuli, all showed a much more extensive involvement for the colored than for the form stimuli. Of the four eyes examined in an advanced stage with all three form stimuli, all showed the greatest involvement with 0.5 degree gray (19 per cent) on black, next with 0.17 degree white on black, and the least with 0.5 degree white on black.

The use of colored stimuli in scotometry is obviously of great advantage. The use of form stimuli of low visibility is also of great advantage. It is difficult, however, to give form stimuli as low visibility as color stimuli and stay within the limits of working feasibility. To give the fields, for example, for a 1 degree white stimulus the same order of magnitude as those for 1 degree red or 1 degree blue, all under 7 foot candles of illumination, would require the use of a background differing from it in reflecting power by only about 11 per cent. This represents a small sensation difference in the part of the scale involved. However, decrease in size and decrease of intensity of illumination can be combined with relation to background as sensitizing factors with results that are as yet unknown. Both decrease of size and of intensity of illumination also can be used with colored stimuli with even greater effect. As we have stated in earlier papers, the only limit to the sensitivity that may be attained is feasibility. In any event, it seems that the omission of the use of colored stimuli for work in scotometry would be most unfortunate.

It may be interesting at this point to list some of the advantages of a tangent screen that can be used at 33 cm. as compared with the more distant screens, more particularly perhaps to make the comparison for the screen which was designed as an attachment to the Ferree-Rand perimeter:

(1) The great ease with which colored stimuli can be used. The use of these stimuli requires a background of gray of the brightness of the color. It is much easier to provide these backgrounds and to give them the proper care for the smaller screen.

(2) The ease with which the devices designed to add sensitivity to the form stimulus can be used. Previously sensitivity has been added only by decreasing the size of the stimulus. Indeed this means for adding sensitivity is presumably the principal, if not the only, reason that the larger and more distant screens have been used. Decrease of size, however, is not the only way in which low visibility can be given to the stimulus and sensitivity added to the examination, as we have

pointed out. Decrease in size, decrease in coefficient of reflection between object and background, and decrease of intensity of illumination are all effective when used singly and still more so when used in appropriate combination. The provision is made for the use of all of these devices singly and in combination in the instrument in question. The reduction in size of stimulus can, we believe, be carried as far as is advisable on the 33 cm. screen. Too great a reduction introduces difficulty in connection with differences in condition of refraction. In proportion as refraction is involved, the test loses its right to be classed as a test of the powers and functions of the retina.[7] In fact, to carry the procedure too far in case of any of these means of reducing visibility involves undesirable features. It seems more advisable to reach the extremes of sensitivity by an appropriate combination of devices.

(3) The ease and precision with which the eye can be placed and held in correct relation to the tangent screen and its graduations. Such means have not as yet been provided for the more distant screens, and they are not nearly so easy to provide in case of these screens.

(4) Accuracy of control of fixation. All the devices that can be used for the control of fixation for work on the arc of the perimeter can be used with equal convenience and effect on the tangent screen. The control of fixation is extremely important in scotometry. Satisfactory fixation devices have not as yet been provided for the distant screens. Moreover, the examiner cannot so readily and satisfactorily watch and control the behavior of the patient with reference to both the location of the eye and the fixation in case of the more distant screen.

(5) Uniformity and constancy in both color and intensity of illumination. By moving the arc of the perimeter into the meridian under examination (lamp arm at right angles) almost as uniform illumination of the stimulus can be attained as is possible on the arc of the perimeter. When the lamp arm is moved with one hand and the stimulus with the other, this precision of control is easily and conveniently maintained.

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7. In this connection it must be remembered that the purpose of the test in perimetry and scotometry is to find the point in the field at which the stimulus in question is of threshold value for the light and color sense. This point is called the limit of sensitivity for that particular stimulus. Such extremely small stimuli as are sometimes used, particularly in scotometry, are more properly test objects for acuity than for the sensory process. In fact just this type of object was used and recommended for testing acuity by Wolffberg, Guillery, Fridenburg, Aubert and Foerster, Landolt and Ito, etc. Strictly speaking, the test object used to determine the limit of light sensitivity in perimetry and to outline the limit of sensitivity in case of a scotoma should not be called a form stimulus, for the discrimination of form and detail is the special province of the acuity judgment.

Any attempt at a satisfactory illumination of a large tangent screen involves both great difficulty and expense.

(6) The compactness of the field of operation. The examiner is in convenient relation with both the patient and the instrument. This saves time and energy and secures much better coordination of effort and facilities.

(7) The tangent screen as an attachment to the perimeter. This entails little additional expense and requires no additional space.

In recent publications, Dr. Luther Peter [8] stated that he has been able to get more accurate and dependable results with the 33 cm. screen provided with our instrument than with the screens of longer radius. While we have never made a comparison of this kind, we are not surprised that such results have been obtained, so much better in general are the working conditions for the nearer screen on all points that are important for precision and reproducibility.

An extreme example of the importance of steadiness of fixation for the detection of scotomas may be had in a case of retinitis pigmentosa which recently came under our observation. The patient's eyes executed searching movements with a range of from 30 to 35 degrees on either side. These movements were more frequent and more pronounced when one eye was closed. The examination of the fundus revealed the characteristic appearance of retinitis pigmentosa. Vision was low, but the patient was not conscious of any unevenness of the field of vision. No success was had in field study with the usual controls of fixation. However, when a high visibility was given to the fixation object a different situation was created. Once visibility was attained in the central field sufficient control was exercised over the searching movements to enable a fairly satisfactory study to be made. For the right eye, sufficiently high central visibility could be attained by using the luminous target of the presbyopic fixation device of the Ferree-Rand perimeter at its highest brightness. For the left eye the lamp of an electric ophthalmo-

8. Peter, Luther C.: Observations on Central, Paracentral, and Caecocentral Scotomas, Tr. Am. Ophth. Soc. **25**:275, 1927; Methods and Results in Perimetry, Canad. M. A. J. **21**:288, 1929. In a discussion of the first of the foregoing papers Dr. T. B. Holloway says (idem. p. 283): "We have several hundred records made with the Ferree-Rand perimeter, and in our scotoma work we have checked our findings with the campimeter attachment of the perimeter, with the screen at one meter, sometimes at two meters. So far, in the majority of cases, the Ferree-Rand instrument has appeared to be the more satisfactory, but in several instances the reverse was true."

scope was used as the fixation object. In the work on the tangent screen large scotomas were found in each eye. These scotomas are shown in chart 18.

In connection with these scotomas, it is interesting to note that the eye, unable to see objects of ordinary visibility in the central field, executed searching movements to bring the images for this field on to the more sensitive peripheral portions of the field. These movements were made so effectively that the patient was not conscious of the gap in her field of vision. Looking at a blank white surface under ordinary working illumination, for example, the surface seemed to her quite uniform in brightness. When the foregoing fixation objects were used, however, the scotomas became distinctly outlined as dark shadows. Two points of special interest may be mentioned in connection with the case: (*a*)

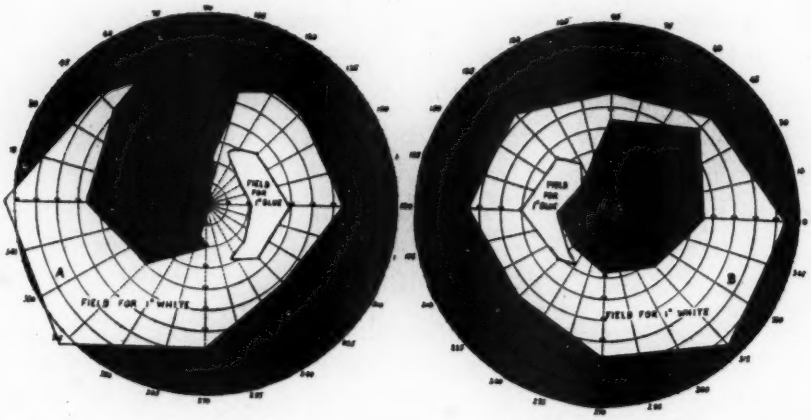

Chart 18 (case 16).—Retinitis pigmentosa. Showing scotomas which were visible to the patient with controlled fixation, but invisible with uncontrolled fixation. The control of fixation needed for mapping these scotomas could be secured only by use of a luminous fixation object of high visibility.

By the use of this extreme example a striking demonstration is made of the great extent to which unsteadiness of fixation interferes with the search for scotomas. With the degree of unsteadiness present in this case every point in the field was effective for vision even though large areas of the retina were blind. (*b*) The case gives a striking demonstration of the great versatility shown by the eye in its remedial devices.

SUMMARY AND CONCLUSIONS

1. Three methods of reducing the visibility of form stimuli for the purpose of increasing their sensitivity, both diagnostic and prognostic, have been proposed: decrease of size of stimulus, decrease of difference

in coefficient of reflection between stimulus and background, and decrease of intensity of illumination. These methods may be used either singly or in combination. The first two have been used with very good results in this study.

2. The use of colored stimuli, however, has proved to be enormously more effective in the detection of incipient scotomas and small changes in the advance or recession of the scotomatous condition. There are two possibilities of explanation of this greater diagnostic and prognostic sensitivity. (*a*) The low visibility of colored stimuli, particularly in the paracentral and peripheral portions of the retina. From the center to an excentricity of 60 degrees, dark adaptation, the retina may have from a little more than 1 to 14,000 times greater chromatic than achromatic sensitivity to spectrum light. (*b*) A possible special susceptibility of the color processes to pathologic conditions.

3. It is difficult to give form stimuli as low visibility for the paracentral and peripheral field as color stimuli and stay within the limit of feasibility. To give the field for a 1 degree stimulus (78 per cent coefficient of reflection) the same order of magnitude under 7 foot candles as the field for 1 degree red or 1 degree blue under the same intensity of illumination would require the use of a background with a coefficient of reflection of about 67 per cent (approximately midway between Hering gray nos. 1 and 2). The sensation difference between stimulus and background in this case is very small indeed, too small for feasibility. Also, decrease in size or decrease in intensity of illumination is so much more effective for color than for form that it is obvious that the decrease of either to such an extent as to give the form stimulus the same reduction in visibility as is possible for color stimuli would transcend the limits of working feasibility. Moreover, in case of color there is the possibility of a special susceptibility to pathologic changes because of the greater instability of the color function.

4. The effective use of colored stimuli requires a background of gray of the brightness of the color. When lighter or darker backgrounds are used, the test object does not disappear as it should for portions of the retina defective to color, but fades into a gray which acts as a form stimulus. On black as a background, most of the colors could be seen as a very light gray or as white when the color disappears, due to physiologic induction or contrast, particularly in the paracentral and peripheral portions of the retina where the physiologic induction is especially great. Also the use of backgrounds differing in brightness from the color gives much more erratic results in any test for color sensitivity.

5. Colored stimuli and form stimuli of low visibility are of great value in diagnosis when the presence of scotomatous areas or enlargements of the blind spot is an important feature in the case. In many cases the use of such stimuli can, we believe, be made to yield the first test evidence of the presence of a pathologic condition.

6. Colored stimuli and form stimuli of low visibility are of great value in prognosis, i.e., in showing the course which the disease is taking. Results are presented in this paper in which for periods, both of advance and recovery, the use of these stimuli alone showed that a change was taking place. The evidence presented by the use of colored stimuli in these cases was particularly striking.

7. The use of colored stimuli and form stimuli of low visibility affords abundance of evidence that what is commonly called the Bjerrum scotoma may first make its appearance as a detached scotoma and later merge with the blind spot.

8. Sensitive means of detection show that in the incipient stages of glaucoma the amount of functional disturbance fluctuates, comes and goes. Negative results at one or more times can therefore not be regarded as conclusive.

9. The scotomatous extension of the blind spot in glaucoma may begin at either its upper or its lower margin. Both types are of frequent occurrence. Also the merging of the scotoma with the blind area produced by the contracting field may come either above or below the blind spot.

10. The indications thus far are that the use of color stimuli and form stimuli of low visibility have little, if any, advantage as compared with 1 degree white on black in a diagnosis based on area or size of field because of the greater range of scatter of their results for normal eyes owing to the higher sensitivity of these stimuli for the detection of individual differences. For scotometry, however, for blind spot work, for the detection of cuts in the field and irregularities in the shape of the field, and for following the changes in the size or shape of the field with delicacy and precision during the advance or recession of a pathologic condition, they have a great and absolutely indispensable advantage.

REFLEX TRANSMISSION OF STIMULI FROM ONE EYE TO THE OTHER

THE "DARK-LIGHT TEST" FOLLOWING NEURECTOMY *

ARYEH FEIGENBAUM, M.D.
JERUSALEM, PALESTINE

"The "dark-light test," first described by Groenholm [1] in 1910 and studied by Seidel [2] since 1920, is an important functional test in glaucoma. Besides its practical interest, it has a special theoretical value. The test consists of three tonometric measurements: the first made before and the second after a stay in the dark, and the third made after illumination of one eye while the other eye is entirely protected from light. Thus a "dark-light curve" is obtained. In 1928, I [3] communicated my experience with the test in a considerable number of cases.

In glaucoma an increase of intra-ocular pressure after a stay in the dark and a decrease after exposure to light are often found. A dark-light curve can also be observed in the majority of normal eyes, although faintly. The reaction is especially characteristic of primary, simple and chronic inflammatory glaucoma with a shallow anterior chamber. Differences in the amplitude of fluctuations are of value in the differential diagnosis of early glaucoma. After illumination of one eye, one observes a striking fact: a parallel lowering of the intra-ocular pressure in the other eye, which is entirely protected from light.

Thus in the dark-light test one has to consider separately two processes: (1) the direct effect of the dark leading to a rise of pressure and the direct effect of illumination leading to a lowering of pressure in the single eye, and (2) the lowering of pressure in one eye after illumination of the other, even if the former remains in the dark. According to Groenholm and Seidel, these changes in pressure are due to the width of the pupil, so that for purely mechanical reasons the rise of pressure in the dark would be due to dilatation, and the lowering of pressure in the light would be due to contraction of the pupil (resulting from obstruction or freeing of the chamber angle); according to this view, the parallel lowering of pressure in the second eye after illumination of the first must be attributed to the consensual pupillary reaction.

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* Submitted for publication, Sept. 16, 1930.

* Read in abstract at the Thirteenth International Ophthalmological Congress, Amsterdam, Sept. 10, 1929.

1. Groenholm: Arch. f. Augenh. **66**:346, 1910; **67**:136, 1910.
2. Seidel: Arch. f. Ophth. **102**:417, 1920.
3. Feigenbaum: Klin. Monatsbl. f. Augenh. **80**:577, 1928.

This explanation, however, must be rejected for several reasons. The first counterargument is derived from experiments on eyes in which the width of the pupils was fixed in various ways (by drugs, such as pilocarpine and homatropine, by aniridia, after iridectomy and by circular synechiae and internal ophthalmoplegia [4]); the second proof arises from the illumination of blind eyes with a consequent lowering of pressure in the second (seeing) eye (charts 1 and 2). Further tests with light on blind eyes show that there are exceptions to this rule, i. e., no influence of the illumination of the blind eye on the second eye, especially when the absolute glaucomatous eye had become hypotonic by degeneration (chart 3). I shall again refer to this fact.

The question of reflex transmission of a stimulus from one eye to the other was investigated (chiefly in order to explain the so-called sympathetic irritation) by Mooren and Rumpf [5] (1880), Gruenhagen and Jesner [6] (1880), Bach [7] (1895) and others, who confirmed its possibility. The negative experimental results, especially those of Wessely [8] (1900) form weighty objections. According to Axenfeld [9] (1909), however, they do not prove that observations made on animals are necessarily entirely valid for the human eye.

In recent experiments Woelfflin [10] (1922) found that repeated irritation of one cervical sympathetic nerve produced slight rises in intraocular pressure on the other side.

Leplat [11] (1924) found that experimental contusion of one eye produced a rise in pressure followed by a parallel rise in pressure in the other eye. It was his opinion that contusion of one eye produces a dis-

4. A rare case, hitherto unreported, of ophthalmoplegia and glaucoma, in which dark-light tests were performed may be of interest: For several years, I had observed a man, aged 40, with microcornea of both eyes. The pupils were immobile for undisclosed reasons (paresis of the lower branch of the right facial nerve was the only other central symptom); both in the light and in the dark the right pupil measured 3 mm., and the left 6.5 mm., in diameter. Bilateral simple glaucoma was present. The left eye (with the narrower pupil) became blind, while the right eye (with a maximal dilation of the pupil!), which showed a contraction of the visual field, retained a vision of 5/12. The patient refused operation. After a sojourn in the dark, the right eye showed regular rises in tension from 51 to 60 and from 43 to 51 mm. of mercury, and the left eye from 24 to 40, from 26 to 51 and from 31 to 51 mm., etc. The falls in tension after illumination were less regular, especially in the right eye; in the left eye, they were from 37 to 26, from 37 to 22, from 41 to 26 mm., etc.

5. Mooren and Rumpf: Zentralbl. d. med. wissensch., 1880, no. 19.

6. Gruenhagen and Jesner: Centralbl. f. Prakt. Augenh. **4**:181, 1880.

7. Bach: Ber. d. 24. Vers. Heidelberg, 1895.

8. Wessely: Arch. f. Ophth. **50**:1 and 123, 1900.

9. Axenfeld: Klin. Monatsbl. f. Augenh. **47**:117, 1909.

10. Woelfflin: Klin. Monatsbl. f. Augenh. **68**:460, 1922.

11. Leplat: Ann. d'ocul. **161**:87, 1924.

turbance in the permeability of the walls of the vessels which allows a transudation of serum-albumin, probably by forced dilatation. In other words, there is a vasomotor sympathetic reflex, which influences the tension at the same time.

Besso [12] (1924) reported five cases of glaucoma in which iridectomy was performed simultaneously on both eyes. Immediately after the operation on the second eye, bleeding occurred in the first eye which had not previously bled. The bleeding filled the anterior chamber partially

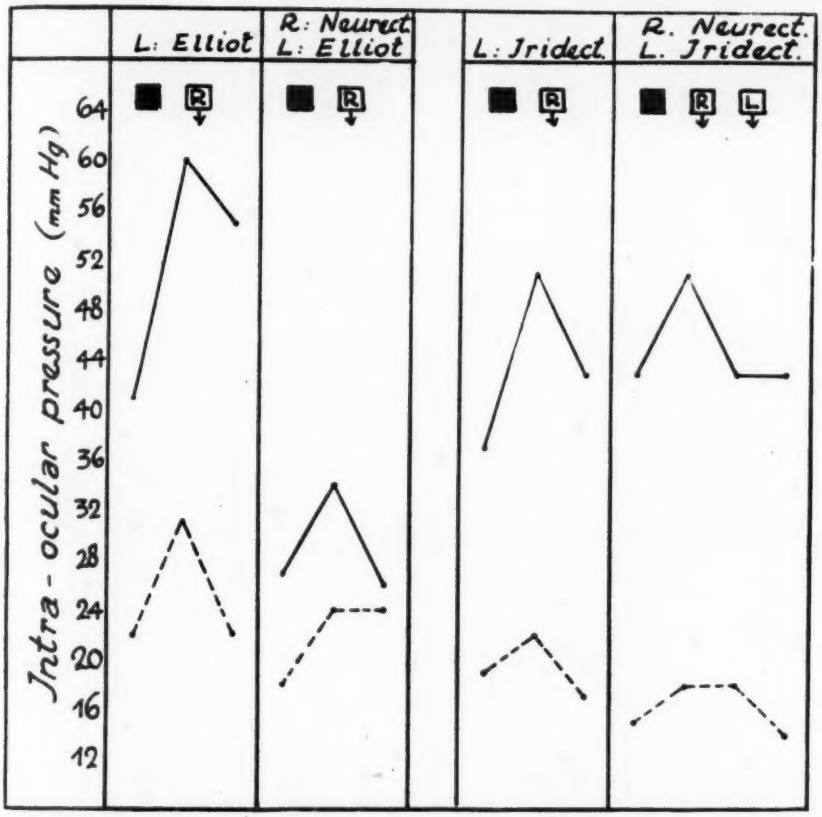

Chart 1 (cases 1 and 2).—Results of a dark-light test before and after neurectomy. The two columns at the left indicate case 1, and the two at the right, case 2. In this and the following charts, the black square denotes stay in the dark and the white square illumination of the right or left eye (*R* or *L*), while the other is entirely protected from light. The solid line indicates the right eye, and the dotted line the left eye.

in some cases and totally in others. To explain this occurrence he assumed that the bleeding which occurred in the first eye after operation on the second was of reflex origin.

12. Besso: Boll. d'ocul., 1924, vol. 3.

Magitot [13] (1927-1928) analyzed a series of symptoms appearing in eyes under certain conditions (after subconjunctival injections, contusions of the eyeball, injuries to the cornea and irritations of the iris and ciliary body). He concluded that short reflex connections exist exterior to the centers in the eye itself and from one eye to the other,

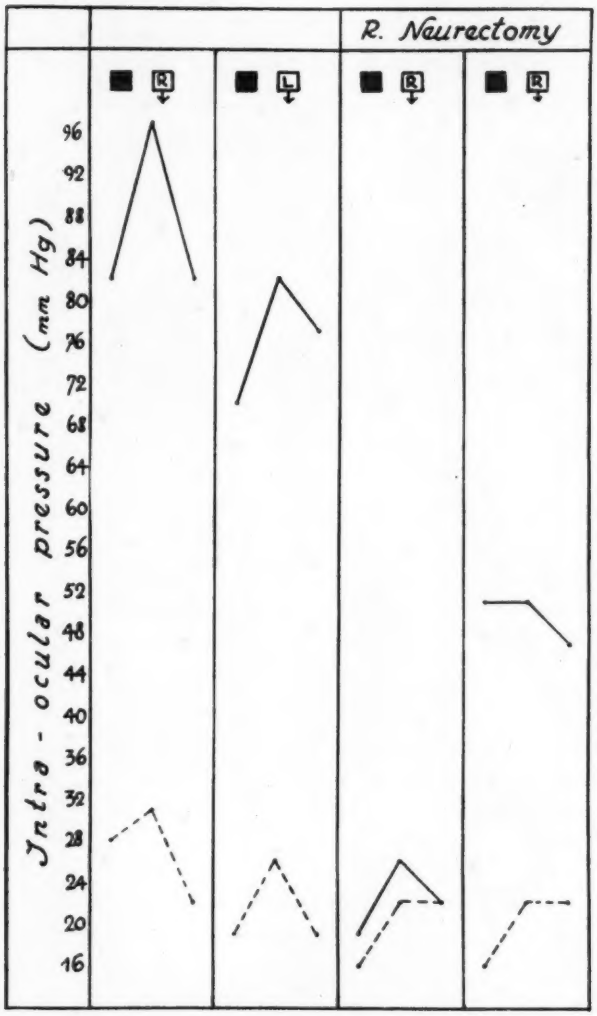

Chart 2 (case 3).—Results of a dark-light test before and after neurectomy.

in short, the so-called axon reflexes of Langley. He adopted the view that in primary glaucoma and in certain related conditions dilatation of capillaries and alteration in the permeability of the vascular walls are principally responsible for the pathologic rise in pressure.

13. Magitot: Ann. d'ocul. **164**:482, 1927; **165**:481, 1928

Shiraishi [14] (1928) compressed the ciliary ganglion on one side in twenty-nine dogs; in twelve animals he found opacity of the cornea of the second eye after a lapse of time varying from fourteen to fifty days. He ascribed this phenomenon to a chronic irritation of the ciliary ganglion of the other side, transmitted from the compressed ganglion.

Hamburger [15] (1928) showed in four cases an inflammatory softening of one eye communicated to the other through the transmission of

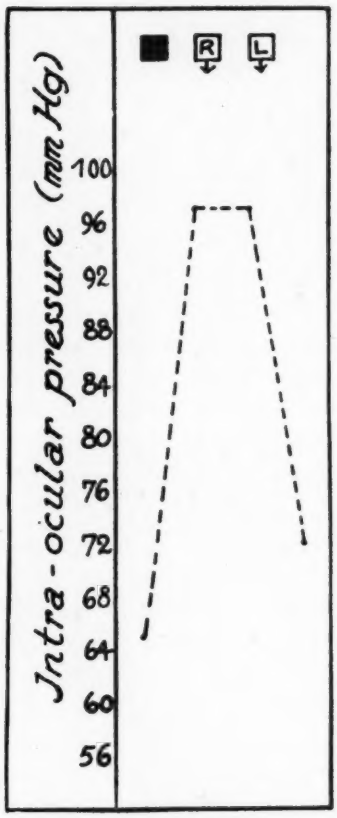

Chart 3.—Results of a dark-light test in the presence of chronic inflammatory glaucoma of the left eye and degeneration of the right eye.

irritation. His graph 3 is particularly noteworthy, because it shows how one eye recently affected with iritis softens and transmits the lowering of tension to the other nonirritated eye, which was practically blind with seclusion and occlusion. This is further proof that the effect is not produced by simple alteration in the width of the pupil.

14. Shiraishi: Arch. f. Ophth. **120**:576, 1928.

15. Hamburger: Klin. Monatsbl. f. Augenh. **81**:620, 1928.

Redslob [16] (1928) injected acid solutions with a high concentration of hydrogen ions into the vitreous of one eye. In the untreated eye a fall in tension occurred. When alkaline solutions were used there was a rise in tension. He concluded that this is the result of a sympathetic vasomotor influence of the altered eye on the untreated one.

Thiel [17] (1929) assumed, from experimental irritation of the sympathetic with its marked effect on the intra-ocular pressure of normal and glaucomatous eyes (first a brief rise and then a longer fall of tension), that there was a reflex action on the vessels of the choroid, transmitted by the sympathetic. A unilateral irritation of the sympathetic produces a parallel reaction in both eyes, from which one may conclude that there is a reflex connection between both eyes.

In 1928 I reported a good example of a reflex process in the eye, probably based on a vasomotor process. After trephining was performed on one eye with chronic inflammatory glaucoma, deposits of cells appeared on the posterior surface of the cornea, at first on the completely unirritated eye on which operation had been performed, and then on the other eye which was likewise free from irritation. The deposits disappeared from both eyes without a trace on the thirteenth day after the operation. The considerable lowering of pressure probably caused an increased transudation of albuminous fluid into the aqueous, and this communicated itself to the other eye by a reflex vasomotor stimulus.

In this connection certain observations and conclusions were recently communicated by Birch-Hirschfeld [18] on the basis of experiences with a considerable series of day-curves of tension in glaucomatous patients. He confirmed the well known fact of the far-reaching parallelism of curves for both eyes even in unilateral glaucoma. After trephining nearly blind eyes he observed such a great lowering of pressure even in the glaucomatous eye that was not operated on that operations on the better eye became unnecessary. The conformity of the tension curves in both eyes indicates that there is an underlying cause which may possibly be explained by a hypothetic directing center (uebergeordnetes Zentrum).

There are series of reflex vasomotor processes in other pairs of organs, e. g., congestion of the blood of both upper extremities (warming of one arm causing a simultaneous dilatation of the vessels of the other arm, according to Leriche and Fontaine) and a reflex anuria that sometimes occurs after extirpation of the kidney on the other side.

A further proof that the dark-light test represents a reflex vasomotor process in the eye would be forthcoming if it could be shown that after interruption of the conduction of stimuli in the first eye the fluctuations in the tension of the other eye cease.

16. Redslob: Bull. Soc. d'opht. de Paris, no. 9, bis, p. 640 (Dec.) 1928.
17. Thiel: Klin. Monatsbl. f. Augenh. **82**:78, 1929.
18. Birch-Hirschfeld: Ztschr. f. Augenh. **70**:1 (Dec.) 1929.

RESULT OF TESTS

For this purpose I thought that experiments on patients with glaucoma before and after an opticociliary neurectomy would be suitable. A dark-light test was carried out several times on three patients with painful chronic inflammatory glaucoma before and after operation on the blind eye. Before the operation all three cases showed typical fluctuations. After neurectomy the tension in the blind eye rose after a stay in the dark and could be lowered through direct illumination, yet a parallel fall of tension in the second eye did not occur after illumination of the first. The result of these repeated tests was characteristic of an interruption of conduction of stimuli from one eye to the other (curves in charts 1 and 2). The cases were as follows:

CASE 1.—Rachel B., aged 50, observed on July 12, 1928, had undergone iridectomy on both eyes about twenty years before. The right eye was painful because of absolute glaucoma with bullous keratitis. The left eye presented a chronic inflammatory glaucoma, normalized by iridectomy (a shallow excavation of the optic disk). Neurectomy was performed on July 16 (chart 1).

CASE 2.—Moshe H., aged 48, examined on Sept. 2, 1928, complained of severe headaches on the right side two months before. He became blind in the right eye under the treatment of a quack. A trephining was performed on the left eye. Because of severe pain in the right eye neurectomy was performed on October 22 (chart 1).

CASE 3.—Malka M., aged 61, seen on Nov. 18, 1928, had absolute painful glaucoma of the right eye. The anterior chamber was moderately shallow. There was chronic inflammatory glaucoma of the left eye; vision was limited to the perception of rainbow tints (chart 2).

Tests made on two other patients on whom neurectomy was performed gave negative results, but in the first case no typical reaction was obtained before neurectomy (a fall of tension did not occur after illumination); the second case was one of unilateral glaucoma secondary to thrombosis of the central retinal vein.

SUMMARY AND CONCLUSIONS

From these tests two conclusions may be reached: 1. The neurectomized eye reacts in a typical way, although the sensitive connections with the centers are cut. Thus one may assume a so-called axon reflex, i. e., a short reflex that does not pass through the center (intra-ocular vasomotor reflex of the uveal vessels). 2. The influence of the second eye probably likewise consists in a primitive reflex of this kind, i. e., an interocular vasomotor reflex transmission of stimuli. It is thus comprehensible that in absolute glaucoma with hypotonia (atrophy of the iris and secondary cataract) in which degeneration of the relative nervous receptor organs has occurred, the stimulus meets with no response (chart 3).

With regard to the aforementioned assumption of Birch-Hirschfeld, and also in neurectomized patients, the results of the dark-light test seem to point more to the probability that in primary glaucoma the parallelism in reaction comes from mutual influence of both eyes, and that the lowering of pressure in one eye, after the trephining of the other, must be ascribed to a vasomotor reflex. It is also easy to understand why a secondary glaucoma often does not show this parallelism, as it frequently arises not from vascular but from coarse mechanical lesions, for instance, from luxation of the lens. In all probability, a certain predisposition of the vessels is a primary condition for the parallelism, somewhat as Schmidt regards the endothelium in patients with glaucoma. This predisposition would then be the hypothetic directing cause for the conformity in the reaction of eyes with primary glaucoma.

In general it may be said that, in the dark, eyes behave as if there were an irritation of the cervical sympathetic (dilatation of the pupil, a rise in tension), while with regard to the effect of light (contraction of the pupil, a fall in tension) it appears as if a parasympathetic stimulation is predominant. The character of the processes that occur in the vessels with regard to constriction, dilatation or alteration in permeability are not discussed in this article.

Finally, in the light of manifold experiences, one should not speak of a causal connection between the width of the pupil and ocular tension, but rather of a far-reaching coordination.[19]

19. In a series of experimental injections of physiologic solution of sodium chloride into the vitreous, von Hofe (Arch. f. Augenh. **102**:315, 1920) found in the treated eye a reactive hypertonia followed by hypotonia. He stated "that up to a certain degree these phenomena manifested themselves as well in the unteated eye." Thus the direct transfer of stimuli from one eye to the other after the unilateral injection of epinephrine, claimed by Hamburger (Med. Klin. **20**:274, 1924), becomes more probable, contrary to the assumption of others that the effect is transmitted by the general circulation of the blood.

EXTENSION OF GLIOMA (RETINOBLASTOMA) INTO THE OPTIC NERVE*

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The fact that an intra-ocular new growth may spread into the optic nerve is well known. For this reason, one is led to secure a long portion of the nerve at enucleation. The following presentation deals with the results of examining globes enucleated for glioma, with the idea of determining the frequency of nerve invasion, the frequency with which the nerves are severed at operation distal to this invasion and the frequency of extra-ocular extensions. For the purpose of comparison, a small series of cases of sarcoma of the choroid is given.

The microscopic examination of 119 eyes enucleated for glioma with no extra-ocular extension showed that in 63, or 52 per cent, the gliomatous tissue had invaded the optic nerve posterior to the lamina cribrosa. Of these 63 cases, 51, or 81 per cent, showed that the optic nerve had not been severed distal to the extension of the tumor. In other words, 43 per cent of the 119 cases were doomed to failure before the patient left the operating room.

It was obvious that the size of the new growth in the globe did not have any bearing on whether or not it extended into the nerve. Sometimes the tumor was small and showed a nerve extension. Extra-ocular extension occurred in only 10 per cent of the eyes enucleated for glioma and then late, in the presence of an extensive growth. This is in contrast to sarcoma of the choroid in which the extra-ocular extension was common (eighteen, or 30 per cent, of sixty-two patients examined) and often occurred early. It was extremely rare for sarcoma to extend into the optic nerve (one, or 1.5 per cent, of sixty-two patients) and then late in the course of the tumor, after extensive extra-ocular extension was already present.

Glioma arises from the nuclear layers of the retina and is a tumor of nerve tissue origin. It therefore appears to have a certain affinity for invading nerve tissue such as the optic nerve. It spreads into the

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* Dr. Ernst Fuchs and Dr. F. H. Verhoeff permitted me to study the cases of glioma in their collection of slides. The 119 cases here reported are from their collections, from those at the Eno Laboratory, New York Eye and Ear Infirmary, and from mine. The cases in the different collections yielded strikingly similar results.

* From the Clinical Reports of the New York Eye and Ear Infirmary, March, 1930.

nerve and completely displaces the optic nerve bundles. This happens so literally at times that the size and shape of the bundles surrounded by their septums are preserved, with the tumor tissue occupying the place of the nerve fibers. The tumor has little tendency to invade fibrous tissue, as evidenced by the fact that when the optic nerve is atrophic the hypertrophied septal tissue appears to be a barrier to invasion of the nerve, and by the fact that glioma spreads through the sclera to the extra-ocular structures only exceptionally (10 per cent) and then late when the buphthalmos with distention and thinning of the scleral walls is probably an important factor.

Glioma usually proves fatal by extension through the optic nerve into the brain. It extends into the nerve and gains access to the subdural space directly or at the site where the central vessels enter the nerve. After reaching the subdural space it apparently grows fluently; the bilateral nature of glioma is sometimes due to its spread from the subdural space around the optic nerve of one eye to the fellow eye.

One of the cases in this series was that of a child, aged 15 months, both of whose eyes were affected. Autopsy showed primary glioma in the left eye and an extension through the nerve to the subdural space and hence around the chiasm to the subdural space and globe of the right eye. As glioma is fatal, usually by extension to the optic nerve and only exceptionally by distal metastasis, and as extra-ocular extension occurs late and in a small percentage of the cases, it offers a good opportunity for complete eradication and cure at operation. That advantage is not taken of this is evidenced by the fact that in 43 per cent of the cases that show no extra-ocular extension, the optic nerve is not cut distal to the invasion of the nerve by the tumor. Many of these cases could unquestionably be cured if the nerve was severed as far back as possible. It is reasonable to expect that the nerve should be from 10 to 12 mm. long or longer, but one of this length is rarely found in the laboratory. In fact, the average nerve is not over 3 mm. in length. In view of the fact that 52 per cent of the cases of glioma with no extra-ocular extension show invasion of the nerve posterior to the lamina cribrosa and that 81 per cent of these cases show that the nerve has not been severed distal to the tumor invasion, Mr. Burchell now employs in the laboratory of the New York Eye and Ear Infirmary the following procedure for globes enucleated with the diagnosis of glioma. As soon as the globe reaches the laboratory the nerve is severed flush with the sclera and "run through" in forty-eight hours as tissue for biopsy. If gliomatous tissue is seen extending through the site where the nerve was cut at operation the surgeon is notified. His choice of procedure then probably rests between further excision of the nerve or irradiation or both.

Hirschberg,[1] in 1869, reported cure in 5 per cent of a series of 75 cases of glioma. Wintersteiner,[2] in 1897, reported cure two years after enucleation in 13 per cent of a series of 497 cases. In 28 cases of Leber's,[3] reported in 1910, and in 46 cases of Adam's,[4] reported in 1911, there was cure in 57 per cent. It is interesting to note that this 57 per cent is the supplement of 43 per cent, the figure for cases that showed tumor tissue left on the proximal end of the severed optic nerve.

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1. Hirschberg, J.: Anatomisch Untersuchungen ueber Glioma Retinae, Arch. f. Ophth. **14**:30, 1868.

2. Wintersteiner, H.: Das Neuroepithelioma retinae, Vienna, Franz Deuticke, 1897.

3. Leber, T.: Die Geschwulstbildungen der Netzhaut, in Graefe-Sämisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipsic, W. Engelmann, 1916, vol. 7, chapt. 15.

4. Adam, C.: Statistiches, klinisches und anatomisches ueber das Glioma retinae, Ztschr. f. Augenh. **25**:330, 1911.

MECHANISM OF SOME CONVULSIVE MOVEMENTS OF THE ORBICULARIS AND OF THE FACE

THE MANNER OF THEIR REMOVAL *

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All physicians are familiar with convulsive movements of the face in chorea, due to cerebral infiltration by organismal products. Another type of convulsive movement of the face of physical origin—due to encephalitic irritation—has been seen during recent epidemics of encephalitis. The mechanism of that condition also is simple. Another type of convulsive movement of the face of physical origin is that of orbicular spasm, in which the eyelid twitches. That movement, again, is due to irritation of the facial nucleus of encephalic origin. The management of a disorder of that kind is described in the textbooks. There is, however, a different type of convulsive movement of the face.

REPORT OF CASES

CASE 1.—A woman, aged 50, was sent to me some years ago by Dr. Heffron of Syracuse, N. Y., because of a grimacing of the face. At the same time her head turned toward the right. She had seen an eminent neurologist in New York, and he recommended that the sternocleidomastoid muscle be sectioned. This was done, but no benefit was obtained. She was later sent to me, and the discovery was made that this movement had nothing to do with a physical disorder, but was a movement directed toward an end, which was not brought about by a disturbance of the sternocleidomastoid muscle, but the whole mechanism of turning the head and pulling the face to the right was involved. The stimulus was found to be psychologic. It was found that the patient had a niece with whom she had been in the habit of walking, the niece always walking to the left of the aunt. The niece was about to be married. So determined was the aunt not to consent to this that she could not help turning her head away from the niece, so painful was the idea of her prospective marriage to one of a class of foreigners who had always caused misery to their wives in the many instances she knew.

CASE 2.—This case was similar in character to case 1. The head of the patient, a young woman, turned because in back of her at work was a woman with whom she was always trying to quarrel.[1]

CASE 3.—A woman from South Carolina was sent to me by an ophthalmologist because she could not open her eyes and had not been able to do so for two years. She could not go on the street, conduct her household affairs or read, and she

* Submitted for publication, Sept. 16, 1930.

1. This case was reported in detail in 1922. Williams, Tom A.: Certain Dyskinesias Irremediable by Operative Measures: The Pathogen and the Management, J. A. M. A. **79**:1200 (Oct. 7) 1922.

could scarcely write. She had a high blood pressure and looked like an old woman. Although I recognized the psychogenic nature of the condition, I was unwilling to undertake treatment because of her condition and age. However, examination showed that there was no serious arteriosclerosis or kidney disease. She was much more intelligent than she looked. First I found out the cause of the disturbance. It had come on during the World War, when she had had great responsibilities and anxieties. Responsibility and anxiety caused the convulsion of the face that made the eyes close so tightly, because when a young girl she had formed the habit of closing her eyes whenever a painful sight or even thought occurred. She tried to abstract herself by not seeing what was before her. This action was a manner of withdrawal. It had become a habit, but had never given trouble until the great distresses of the World War. Then the eyes remained closed. At the same time the vision began to age, so she went to an ophthalmologist for the fitting of glasses.

But the constant closing of the eyes produced an effort to keep them open, which led to spasmodic movement. So she was sent to another ophthalmologist, who told her that the glasses she had were wrong and gave her others. Naturally, no good was done, because the trouble was not in the eyes but in the mind. So her condition became worse and worse. Explanation and reeducation enabled her to go home in a month vastly improved; she was able to read and write and go out alone.

CASE 4.—A man from Wilmington tried to keep his face straight by chewing his lips. He was a maritime captain, who during the World War had had great responsibilities. The tic progressed to the eyes also, so that he could not open them and had to be led through the streets. He, too, was vastly improved after treatment.

CASE 5.—A man from Washington could not open the eyes. During my absence in France, he was seen by an ophthalmologist, a neurologist, a psychiatrist, an internist and a serologist, who made various examinations, but found nothing objective.

When I saw him, he was in a state of phobia in reference to his eyes. He feared that he was going to lose his sight, and was so terrorized by his visits to different physicians and hospitals that he could not stop thinking about his eyes. The phobia on which the whole matter really depended had been reinforced by this injudicious treatment, which was not based on knowledge of the case, followed by attempts to open the eyes by using "Will Power." Hence it was necessary sedulously to avoid even examining the patient after the first dignosis had been made. Even visits were studiously neglected as one of the means of persuasion that there was not any condition demanding surgical or medical procedure. This precaution enabled a cure to be obtained in a few weeks in this case of palpebral tic or hysterical ptosis.

The condition had been greatly aggravated by the frequent repetition of examinations, ophthalmologic, psychiatric, medical, neurologic, otologic, serologic, endocrinologic and others. None of these had sufficed to ascertain the genesis of the tic, which was readily done by an intelligent anamnesis in conjunction with a study of the actual character of the ptosis. The falling of the lids occurred irrespective of fatigue, in the absence of conjunctival irritation, and without the least sign of facial paralysis or any other organic nervous disease. Although the closing of the lids was usually imperative, they could sometimes be opened spontaneously by different devices.

This is characteristic of every tic. However, the more the patient wished to open the eyes, the more tightly they closed, and the longer they remained closed.

At first it was only by methods of distraction that I could make the patient open them. These methods, of course, were used merely for diagnostic purposes; therapeutically, they are inefficacious. The patient had already discovered this after the eventual failure of such methods as clacking the tongue, coughing, whistling, etc.

The genesis was interpreted as follows: The conjunctiva had become irritated by considerable driving along dusty roads in an automobile in the glare of the summer sun, in conjunction with the short sleep and the conviviality entailed by late hours. The protection of the eyeballs by lowering of the lids was the consequence. In the manner of the tics, this physiologic response eventually became a psychologic habit. The patient might have shaken off this habit as he had done previously with other tics, but in consequence of repeated medical opinions expressed during six weeks by distinguished physicians, there was added to the habit the phobia of the inability to open the eyes and the fear of loss of sight, and, with it, earning capacity.

This patient, who is subject to frequent stresses, has not had a relapse after ten years, despite overindulgence in alcohol.

CASE 6.—This case was much more difficult to diagnose. In a traumatized woman, although a roentgenogram of the neck showed no displacement of the vertebrae, there could be felt a distinct thickening on the side of the spine toward which the head was turned over the transverse process of the left third cervical vertebra. Accordingly, I rejected the diagnosis of neurosis, believing that I was dealing with a trauma of the neck. The treatment prescribed was manipulation by a skilled operator. The result was that the patient slept that night without the aid of the hypnotic she had been taking. Within a week she was completely free from pain, and could move the neck in every direction. She has remained well for six years, although manipulations are occasionally continued as a precaution.

COMMENT

These cases illustrate the fact that convulsive movements of this kind may depend on psychologic mechanisms even when they originate in an uneasiness of physical origin. There may be some general toxic condition, like Bright's disease and high blood pressure, or an endocrine disorder, as dysthyroidism or dyspituitarism. These conditions make the patient uneasy, and he changes his position. On the other hand, the cause of the convulsive movement may be purely psychologic, worry, anxiety, etc. The physician should determine the mechanism of the movement, so that he can devise methods to overcome it, when possible. This is eminently possible in the psychogenic cases, provided the patient is given an intelligent understanding of himself and the proper training is then followed. It is the process of "conditioning" comprised in psychotherapy.[2]

Convulsive and intemperate tics are accompanied by consciousness of the act, are preceded by a desire sometimes amounting to a passion

2. Williams, Tom A.: Mechanism of the Psychoneuroses, Am. J. Psychiat. **4**:431, 1925; Prevention of Suicide, Am. J. Insan. **71**:559 (Jan.) 1915; Dreads and Besetting Fears, Including States of Anxiety, Their Causes and Cure, Boston, Little, Brown & Company, 1923, pp. 202.

to perform it, and are followed by a feeling of relief. The victim feels compelled to make the movement comprising the tic; hence, the term impulsive or compulsive neurosis. The movements always represent, however incompletely, some voluntary act, e. g., turning the head, wrinkling the nose, shrugging the shouders, biting the cheek, blinking the eye, sniffling or jerking a limb. The end to which the movement was first directed, however, has often passed from the recollection of the patient, and the act itself has often degenerated into a caricature of what it originally was. A simple example is winking of the eyes which has continued for years in spite of the fact that the irritation of the foreign body which first excited it has long subsided. A tic often originates in an idea, which ultimately becomes ignored or forgotten by the patient.[3]

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Ophthalmologic Review

THE RELATION OF THE PARANASAL SINUSES TO OCULAR DISORDERS

ESPECIALLY TO RETROBULBAR NEURITIS *

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There are certain phases of the relationship between the eye and the paranasal sinuses which are apparently so well understood as to require little or no mention. I take it that when one sees a case of frank orbital cellulitis, the sinuses are suspected and the patient is referred to the proper authorities if nasal examination and roentgenograms offer evidence of infection. It has been my experience in such cases that when the sinuses are incriminated, the infection is usually a purulent ethmoiditis or sphenoiditis which leaves no doubt of its etiologic importance, and that in the other cases some cause for a metastatic infection can be located elsewhere in the body.

That the sinuses are foci of infection causing uveitis, episcleritis and deep keratitis is also, I believe, kept sufficiently in mind by most ophthalmologists. The association is not nearly so frequent as one would expect from the frequency of sinusitis in this climate. Gilbert found 4 per cent of his cases of uveitis to be due to disease of the sinuses. Bulson arrived at the same figure, while Brown and Irons found only 2 per cent, and Newton 8 per cent, in their series of cases of iritis. Of a series of 118 cases of uveitis under my observation, 6.8 per cent seemed to be due to sinusitis.

The most interesting phase of this subject, I believe, is the relative importance of the paranasal sinuses in the causation of retrobulbar neuritis. In spite of the floods of ink that have been spilled on this question during the past ten years, it is still decidedly a question, and may be resolved into three principal parts: In what percentage of cases of retrobulbar neuritis is sinusitis the cause? Can retrobulbar neuritis be caused by disease of the sinuses giving no evidence on rhinologic or roentgenologic examination; if so, what is the pathology of the disease of the sinuses? In the presence of retrobulbar neuritis with no apparent cause and with no nasal evidence of sinusitis, should one advise exploratory operation on the sinuses?

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The first question has received widely varying answers. In 4 of 28 cases, Herzog found definite sinusitis, while results of nasal treatment in 12 others led him to consider about half as due to disease of the sinuses. Meller found 4.5 per cent which he considered rhinogenic among cases observed in Vienna during two years. Lange found 3.5 per cent, Heine from 3 to 6 per cent and Langenbeck 3.5 per cent. Scheerer and Maier reported on 203 cases from the Tübingen clinic seen during eight years. They found definite multiple sclerosis as the cause in 66 per cent, suspected multiple sclerosis in 5.5 per cent, over-indulgence in tobacco and alcohol in 5.5 per cent and sinusitis in only 1.5 per cent. In 11 per cent no cause could be found. Weill, in 35 cases, found 18 due to multiple sclerosis, 2 to tumors of the base of the brain, 1 to diffuse encephalitis and 1 to ependymitis. Only 1 case showed ethmoiditis and in this instance operation had no effect. In his experience few cases showed improvement following operation on the sinuses. Redslob, with material from the same city (Strasbourg), was convinced of the importance of sinusitis in retrobulbar neuritis, as were other French observers, including Worms and Rollet.

In America, following the work of Sluder and Wright and the anatomic studies of Loeb and Schaeffer, a great deal of attention was paid to the sinuses. After Sluder described hyperplastic ethmoiditis and sphenoiditis, the rhinologist was no longer able to give a negative report when pus and polyps were not found in the nose, and the frequent association of the triologic specialties with ophthalmology made a consideration of the sinuses natural in cases of retrobulbar neuritis. It seems probable, also, that the large number of relatively young physicians with large private practices was partly responsible for the feeling that something radical must be done as soon as the patients were seen. A few dramatic results, such as often occurred, supported this course, while the possibility of a spontaneous recovery like that often seen by Weill, Scheerer and others, was apt to be disregarded. It was easy, under these circumstances, to fall under what Kalt has called a "therapeutic illusion."

White stated that his first seventeen cases were all considered as due to sinusitis, while later experience led him to consider the sinuses as only one of the foci of infection to be considered, and a relatively unimportant one. In sixty recent cases, he considered the teeth and tonsils to be responsible for 70 per cent (?), while only six showed ethmoiditis and most of these had other foci which were also considered important. The results of treatment directed to such foci were as good as those in his earlier series; 50 per cent of the patients regained normal vision and 25 per cent improved, while the condition of the others remained stationary.

While not many Americans have reported such large series of cases, Le Jeune, Bordley, Peter, Oliver and Crowe, Stark, Crane, Fitzgerald and many others reported cases in which the etiologic importance of sinusitis for optic neuritis was considered as proved; the impression was gathered that most of these authors considered it one of the most important causes of the condition. Cushing went to the opposite extreme, apparently doubting the rhinogenic origin of any of these cases. It is doubtless true that he had seen many patients with tumors of the brain who had recently undergone radical intranasal operations, and also that papilledema is a rare result of sinusitis. It is not, however, as he said, unbelievable that papilledema can occur from sinusitis without increased intracranial pressure, since any inflammation involving pressure on the central vein can bring this about. Peter reported a case in which elevation of the disk of 3 diopters subsided when the sinuses were opened; Ellett's case was similar.

Of thirteen cases seen by myself and associates during the past two years, multiple sclerosis was considered to account for three and purulent sinusitis for four. The remaining six cases must be considered undetermined. A nasal origin was considered probable in most of these, and all but one responded to nasal operations or treatment with improvement in vision, although in one case nasal examination showed no disease, and in the others no definite sinusitis. Such a series shows how inconclusive any opinion would be without more exact records of nasal disease than were to be found in our records.

Reasoning that if optic neuritis were frequently caused by sinusitis, retrobulbar neuritis should develop in a fair proportion of cases of sinusitis, a number of observers examined patients with sinusitis for signs of involvement of the optic nerve, such as enlargement of the blind spot and central scotoma. Markbreiter found that 70 per cent of a series showed defects of the fields. Bordley found enlarged blind spots in 31 of 102 cases, Herrenschwand in 18 of 150 cases, Beck and Pillat in 6 of 53 cases of purulent sinusitis, 4 of 65 cases of nasal anomaly producing poor ventilation, and in 8.5 per cent of 47 normal persons. Van der Hoeve found changes in practically all cases of posterior sinusitis. Herzog examined 500 patients and found slight changes (chiefly an enlarged blind spot) in 9.4 per cent; definite signs which he considered significant of disease of the nerve were found in only 2.6 per cent. In proportion to the number of cases, these definite signs were at times as frequent in acute as in chronic sinusitis. He concluded that while the slight changes in the field reported by some observers were probably not indicative of involvement of the nerve, involvement does occur in a considerable number of cases without being noticed by the patient. While most of these defects disappear

as the acute sinusitis clears up, some nerves remain permanently damaged, and this perhaps explains a certain number of cases in which unexplained optic atrophy is discovered later.

There are several reasons why these opinions as to the frequency of rhinogenic retrobulbar neuritis are at variance, and why a review of them appears to cause nothing but confusion. One of these is that many observers consider recovery of vision after a nasal operation as definite proof that an infected sinus was the cause of the disease, while others who have watched such cases clear up spontaneously insist that in many cases in which operation was performed, the condition would have cleared up without this and they refuse to consider sinusitis as a cause without gross evidence of disease of the sinuses. Another reason for confusion is the failure of one group of observers to have in mind the frequent course of multiple sclerosis, which often produces marked loss of vision as its first symptom. This may clear up completely, either with or without operation on the sinuses, and no other symptoms may develop for years, often not for ten years according to Scheerer. I remember two cases which illustrate especially well how easily these cases may be erroneously considered as due to sinusitis. In a man, aged 31, vision in the right eye was reduced to 20/200, his first trouble dating from five days before observation. A central scotoma and contraction of the field were found, and roentgenograms showed a doubtful condition of the right sphenoid and ethmoids. These were drained, and twenty-five days later, vision had returned to 20/20. Eleven months later it had diminished again to 20/70. Nasal examination gave negative results, but the spinal fluid contained 15 cells per cubic millimeter and an increase of globulin and gave a gold curve suggestive of organic disease of the central nervous system. Vision remained the same under treatment by a neurologist. A young woman with an exactly similar history had the ethmoids drained at the advice of my father, and regained normal vision. Five years later, a neurologist in another city wrote that she was under treatment for definite symptoms of multiple sclerosis, and that vision had failed considerably.

The opposite point of view, however, that of considering all cases of retrobulbar neuritis in which empyema of the sinuses cannot be demonstrated as due to multiple sclerosis, even in the absence of all other signs pointing to it, may prove to be equally erroneous. Thus it will be seen that the percentage of cases attributed to sinusitis in the various series quoted is very much a matter of the observer's personal opinion.

PATHOLOGIC PROCESS

Which of the opinions stated is to be accepted will depend on the answer to the following question: What is the pathology in retrobulbar neuritis caused by disease of the sinuses? Here one meets with a great

deal of theorizing and a few valuable studies, chiefly by rhinologists. White reported on the pathologic process in nine cases in which the tissue was examined by Wright. He found edema, fibrous hyperplasia and chronic inflammatory changes in the mucosa, which sometimes involved the bone. In some cases he considered this to be evidence of an inflammatory process involving the nerve by direct extension. Later experience caused him to change his opinion and to consider the changes found in the tissue too slight to support such a theory. In 1928 he stated: "I have never seen a case except in malignant disease of the sphenoid, where I thought there was direct extension from the sinuses to the optic nerve." He believed that infection occurred almost exclusively by the hematogenous route. Hence he considered infection of the antrum as important in its relation to the nerve as infections of the ethmoid, which he rarely found in his later cases. He considered a few cases due to hyperplasia of the turbinates, with obstruction to the ostia and a resulting negative pressure within the sinuses which favors absorption of toxins and bacteria from the blood stream. O. Beck in sections of the sinus wall or turbinates in 4 cases found a catarrhal inflammation of the mucosa, which was infiltrated with eosinophils and plasma cells. There were no signs of acute or purulent inflammation and he believed the changes should be interpreted as the result of negative pressure in the sinuses producing a type of inflammation which may cause damage to the optic nerve. Bordley's experiments seem to me to offer valuable evidence in the same direction. He placed tampons over the sphenoidal ostium in eight cases of sphenoiditis with normal blind spots. In two cases there were changes in the fields following this procedure: In one, a marked increase in the blind spot developed, and five hours later there was a relative scotoma which cleared up when the tampon was removed. J. Beck examined tissue in a number of cases, and found a noninflammatory rarefaction of the bone. Oliver and Crowe reported ten cases in which the tissue had been examined. While a few showed gross nasal disease, in many instances no discharge or polypi were to be seen, and roentgenograms were negative. These cases showed infiltration of the mucosa and submucosa with eosinophils and plasma cells. Bony changes were not described, and evidently the bone was not sectioned in most cases. These cases were all considered rhinogenic, after careful exclusion of other causes, and with the nasopharyngoscope the authors observed hyperemia surrounding the ostia of the sinuses in all instances. They advised an examination of this sort before cocaine or epinephrine was used, as the use of these drugs conceals this important sign. Redslob showed sections from a child who had died of bronchopneumonia; extension of a purulent sphenoiditis through the bone to the nerve could be seen. The few other postmortem observations

similar to this were chiefly in cases of destructive purulent sphenoiditis. In Pickworth's case a perforation through the sella allowed invasion of the nerve, with cocci demonstrable in its sheaths. Neither sphenoiditis nor optic neuritis was evident during life. Ten Doesschate reported extension from a purulent sphenoid to the nerve in a fatal case of pneumonia; the sections contained diplococci (de Kleijn and Gerlach referred to what was perhaps the same case).

While these cases demonstrate the possibility of direct extension in severe purulent sinusitis, they seem less important for the pathogenesis of the usual case than another case observed by ten Doesschate. In this case, also one of pneumonia, acute rhinitis and enlargement of the blind spot had been demonstrated during life; autopsy, ten days later, showed thrombosed veins near the cavernous sinus and edema of the sheaths of the optic nerve. Coffin observed at autopsy thrombosis of the veins in the ethmoidal wall, orbit and nerve.

If it were necessary, these cases offer definite proof that infection of the nerve sheaths can occur by direct extension through the bony wall of an infected sinus. I think that it is admitted by even the most conservative writers that this happens in occasional cases of purulent sinusitis. Herzog tried to show that it may occur in a larger number of cases, some with negative nasal and roentgen indications. He sectioned the middle turbinate and portions of the ethmoidal walls removed at operation in a number of such cases. He first studied the normal histology of the region, and described the intimate relationship between the dural sheath of the nerve and the cortical spaces of the bone separating it from the ethmoid and sphenoid cells. In twelve cadavers, he found all stages between a type with dense bone having few cortical spaces and a less common type with thin cancellous bone, the cortical spaces of which offer free communication between the submucosa of the ethmoid and the nerve sheaths which form its periosteum and send processes directly through these cortical spaces to the submucosa. He believed that the rarity of this type accounts for the rarity of optic neuritis in comparison with the frequency of sinusitis. In the cases of optic neuritis in which relief was obtained by nasal operations, he found small celled infiltration of the submucosa, with many enlarged vessels, while the bone showed edematous cells with the formation of new bone, and the cortical spaces were congested and filled with fibroblasts and lymphocytes—the picture of chronic productive osteitis. In several cases in which a nasal operation was not followed by improvement, he found the walls of the sinuses macroscopically and microscopically normal. "It must be our duty," he stated, "to explain the histopathology of rhinogenic disease in optic neuritis," a suggestion which I believe cannot be too strongly emphasized. His studies led him to believe in

the direct extension of the inflammatory process to the nerve sheaths through the vessels and cortical spaces of the sinus walls. The effectiveness of his "Daueranämizierung," he believed, rests on the reduction of this edema and inflammation in the bone, and on the hastening of the absorption which takes place spontaneously in most cases of acute sinusitis, as in acute rhinitis.

One of the few attempts to produce retrobulbar neuritis was that of Pichler. He injected dyes and bacteria into the antrum of rabbits and produced pansinusitis but no involvement of the nerve. Dyes stained the bone but not the nerve sheaths.

Meller, in his review of the subject in 1924, emphasized the importance of the pathologic process that Herzog described, and he quoted Neumann's opinion that the slight temporary nasal inflammations following coryza are more apt to produce optic neuritis than is chronic sinusitis with the production of polypi and other gross nasal changes. Hirsch described his own histologic observations in five cases, in four of which changes much resembling those described by Herzog were observed. He considered that this, with other histologic studies, justified the belief that nonpurulent, catarrhal sinusitis may cause retrobulbar neuritis and probably, by direct extension, inflammatory edema of the nerve sheaths.

These studies of Herzog, Hirsch, Oliver and Crowe and others offer valuable evidence that this process may occur. How often does it actually occur, with relation to the frequency of retrobulbar neuritis? In other words, in how many of the cases in which no definite etiology is found does it occur? This will not be known until a large number of cases have been studied more carefully, both histologically and clinically. In looking back, one is amazed at the amount of tissue that has been removed from the nose and simply discarded in doubtful cases. Such tissue, if properly studied, might offer evidence of value for the further treatment and prognosis of these cases. Negative observations will encourage further search for possible intracranial causes or a prognosis that includes the possibility of future multiple sclerosis. A no less important part of this study is the closer cooperation of ophthalmologist and rhinologist, not only before, but after, any operation on the nose. The observation of Oliver and Crowe that hyperemia was to be seen around the ostia with the rhinopharyngoscope in all their cases suggests the great importance of having such observations made known to the ophthalmologist in every case, as well as having a careful report on the condition of the mucosa at operation. The suggestion does not seem radical that in some doubtful cases resection of the tip of a middle turbinate for biopsy might afford information that would guide further treatment.

INDICATIONS FOR OPERATION

What one's attitude will be toward operation on sinuses that are only suspicious in the presence of retrobulbar neuritis will depend on one's conclusions from these studies of pathogenesis. First of all, the other causes of the neuritis must be carefully excluded. It must be admitted that multiple sclerosis causes at least half, and probably more, of the cases. A careful neurologic examination should be made in every case, since the finding of one other sign, such as the loss of abdominal reflexes, would offer good evidence for a diagnosis of multiple sclerosis. This examination should include lumbar puncture, since, according to Stewart, 70 per cent of cases show a characteristic colloidal gold curve, and 40 per cent an increased cell count. Acute myelitis, which is perhaps an acute form of multiple sclerosis, must also be considered, although it is not common. A boy, aged 16, apparently in good health, suddenly lost all perception of light in one eye. The fundus was normal, and neurologic examination yielded negative results. A week later, while attending a baseball game, he fell and was carried home, having complete paralysis of the lower extremities. Under a course of injections of foreign protein by a neurologist, he regained full use of his limbs and normal vision in three weeks. Dollfus reported a case similar to this. Ellett's case, with a fatal outcome from intercurrent sepsis, was of the same nature. Cushing insisted on the frequency with which lesions of the optic nerve resulting from tumors of the brain are taken for rhinogenic optic neuritis. Weill and Nordmann reported several similar cases in which the diagnosis was particularly difficult. In one a unilateral central scotoma gave place, after two months, to bitemporal hemianopia, and roentgenograms confirmed the presence of a pituitary tumor. All the modern means of diagnosis are none too complete for proper classification of these cases.

One of the great difficulties is to obtain all this information without the loss of much valuable time. These cases should be regarded as emergencies, and the various examinations should be made at once as a matter of routine, without waiting for each successive report.

When cases of multiple sclerosis, tumor of the brain and amblyopia due to tobacco and alcohol and the small number of cases showing definite purulent sinusitis are eliminated, from 15 to 20 per cent of cases will remain in which nasal and roentgen observations are negative and in which no other cause is found. In these cases a delay of a week or longer would seem to be accepted by most authorities as proper, since it is during this time that many patients begin to improve spontaneously. This week need not be spent in waiting alone. Sweats and injections of foreign protein have been credited with many good results, especially when multiple sclerosis is considered a possible cause. Since the reports of Herzog and von Herrenschwand appeared, the use of "Daueranämi-

zierung" has been widely tried with results, in many cases, as brilliant as those of operation. Whether or not disease is demonstrated in the nose, a trial of this method would seem to be indicated. White's use of a 3 per cent ephedrine spray every two hours probably accomplishes the same result. Herzog's procedure consists in packing the region of the ethmoidal and sphenoidal ostia twice daily with gauze soaked in epinephrine, the pack being left for two hours. Von Herrenschwand reported thirteen cases in which good results were obtained by this means alone, and Mylius and others confirmed his reports. In three cases with symptoms of multiple sclerosis Herzog saw no effect of the treatment. He used the method for diagnosis in doubtful cases, believing that only rhinogenic cases respond to it. He mentioned a case in which vision failed twice after the treatments were stopped, and in which the condition cleared up permanently after the third course of treatment was instituted. When failure occurs after a primary improvement, he believes that opening the sinuses or improving their ventilation by turbinectomy is indicated.

When no improvement occurs under general and local treatment, the time to wait before making an exploratory operation will vary according to the degree of visual defect. If the visual defect is great and blindness threatens, I believe that most ophthalmologists would advise operation after a week of observation; some would advise it sooner. White expressed the belief that 50 per cent of patients recover spontaneously, 30 per cent require submucous resection or turbinectomy and about 20 per cent require free opening of the ethmoid and sphenoid cells. I doubt if White's dictum that only cases with optic foramina smaller than the normal diameter of 5.35 mm. usually require operation has been generally followed. In spite of his interesting observation that most patients with optic neuritis have small foramina, if one's own nerve were threatened, it is doubtful if one would allow a difference of 1 mm. in the size of this canal to determine the procedure to be followed. The time limit during which operation offers some hope is uncertain. As long as atrophy of the disk has not taken place, there is enough chance to justify operation when nasal disease is suspected; some patients with considerable atrophy have recovered good or even normal central vision.

SUMMARY

1. In the causation of retrobulbar neuritis, multiple sclerosis is the most important disease, probably accounting for over 50 per cent of cases.

2. Cases due to purulent sinusitis comprise about 3.5 per cent.

3. Of the 15 or 20 per cent for which no cause can be found, opinions differ as to how many are caused by latent or hyperplastic sinusitis.

4. There is pathologic evidence that such forms of sinusitis may cause retrobulbar neuritis by direct extension.

5. Before operations on the sinuses are performed in such cases, every effort must be made to eliminate other causes, especially multiple sclerosis and tumor of the brain. Examination of the spinal fluid with reference to the gold curve and cell count should be made in every case.

6. Tissue removed from the nose should be examined histologically in every patient operated on, since it is only through knowledge of the pathologic process in such cases that a better understanding of the whole problem may be obtained.

7. With present information and methods of diagnosis, there will still be a number of cases in which exploratory operation on the sinuses is indicated.

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Correspondence

To the Editor.—This is in reply to your letter of December 6, regarding the history of the "subconjunctival tunneling."

The procedure with the keratome was conceived by me in January, 1905; I had not previously seen any ophthalmic surgeon perform this operation, nor had I seen anything in the literature regarding it. I communicated the procedure in Heidelberg in 1906 (fig. 5), and in *Annales d'oculistique* in May, 1907 (figs. 4 and 27, pp. 351 and 365).

One of my predecessors, C. Bader (1881), made an extralimbal incision upward with an ordinary Graefe cataract knife, leaving the conjunctiva uncut; thus the iris—and often the processus ciliaris—fell out subconjunctivally (On Sclerotomy, *Tr. Ophth. Soc. U. Kingdom* **2**: 127, 1881-1882).

Another of my predecessors, H. Herbert, in India, made a short limbal incision and extralimbal conjunctival flap with a narrow (1 mm. or tapered) Graefe knife, performed iris inclusion and then centrally, for the inclusion, a narrow iridectomy (*Tr. Ophth. Soc. U. Kingdom*, 1903).

I believe that most ophthalmic surgeons who try iridencleisis and meridional iridotomy—formerly like iridencleisis an exclusively optical operation—make ordinary conjunctival flaps, often (wrongly) without subsequent conjunctival suture. A suture should never be placed in the included iris (as in G. Critchet's optical "iridodesis" [1858]).

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CORRECTION

An error occurred in the first sentence of the abstract of Puscariu's article, entitled, "Cataract in Diabetes and Treatment with Insulin," from *Soc. Român. de oftal., Bucuresti* **11**:34 (Nov.) 1929, which appeared in the December, 1930, issue of the Archives of Ophthalmology, page 946. This sentence should read:

"A girl, aged 16, with diabetes of three and one-half years' standing and cataract in both eyes of six months' standing, was very weak, having 4.5 mg. per hundred cubic centimeters of blood and glycosuria of from 150 to 180 Gm. in twenty-four hours."

Obituaries

ERNST FUCHS, M.D.
1851-1930

With the death of Prof. Ernst Fuchs, international ophthalmology loses one of its greatest figures. He had so successfully escaped the ills of advancing years, retaining a remarkable mental vigor and interest, that it is hard to realize that he has passed on, and that so many have now lost a much respected friend and an always ready guide.

Born in Vienna, in 1851, he died in that city on Nov. 21, 1930, of angina pectoris, after a brief illness. Fuchs received his medical education in Vienna and was a special pupil of Brücke, Billroth and Arlt. After graduating in 1874, and acting as assistant to Arlt from 1876 to 1880, he rapidly rose to a position of distinction, and became professor of ophthalmology at Liège at the early age of 29. In 1885, he returned to Vienna to succeed Jaeger, and he remained as professor and director of the eye clinic until 1915, when he retired because of the age limit.

During these years, the "Klinik Fuchs" became far-famed and the mecca of all young ophthalmologists; there is scarcely an oculist of importance in this generation who has not profited and been inspired by the teachings of this great scientist.

There in the rooms of the II Augenklinik, one flight up over the entrance to the old Krankenhaus in the Alserstrasse, every morning, after the visit to the wards, Fuchs rapidly passed in review the many new patients, quickly sorting them into groups and selecting those suited for the daily lecture clinic. The fame of the clinic was so well established that patients from all over the world thronged there, and nowhere could such a variety of ocular diseases have been seen and studied.

Into a small notebook that he always carried with him were carefully jotted down in shorthand, new and striking conditions. An unusual memory and a keen power of observation enabled him to recognize and give the descriptions of the many new clinical conditions that carry his name, such as dystrophia epithelialis corneae, episcleritis periodica fugax, keratitis punctata superficialis, keratitis disciformis, Fuchs' macular spot in myopia, retinitis circinata and others. But it was in his clinical lecture that his intellectual powers rose to their highest, and he exerted a lasting impression on his many listeners. With a remarkably clear and logical approach, each case was described and elucidated in

the simplest language without any controversial discussion. His unusual ability as a draughtsman enabled him to illustrate still further the subject under discussion by a quickly executed crayon drawing. One morning of the week the lecture consisted in a demonstration of projected microscopic slides illustrating the lesions studied in the preceding days.

Fuchs gave little of his time to private practice, realizing the obligation that he owed to the university and to the hospital.

His greatest achievement, however, was his "Text Book on Eye Diseases," which has been a classic in German for many years, and which has appeared in numerous editions and translations. It is written in the same clear, straightforward way that characterized his lectures, and has contributed more than can be described to the knowledge the ophthalmologist of the present day possesses. The admirable translation by Duane made his textbook the favorite among English-speaking practitioners.

Another field in which he so industriously worked is that of the pathologic anatomy of the eye. As Meller states, it was Skoda's example that led Fuchs to combine pathologic studies with clinical observation, and to follow the ideal of recognizing in a diseased condition the underlying pathologic changes in the tissue. In this connection may be mentioned the studies of intra-ocular infections that he called endophthalmitis septica, and it was Fuchs who first brought light on that baffling problem, the pathology of sympathetic ophthalmia.

After the World War, so disastrous to the central powers, he, with so many others, had lost everything except the house he lived in. Having retired from active practice, he accepted an invitation to deliver in Spanish lectures on the pathology of the eye in Madrid, and these lectures were then repeated in several cities in North and South America.

A great linguist, an enthusiastic Alpine tourist in his earlier years, his love of travel brought him to most parts of the world. He came repeatedly to this country, where he had many friends and admirers. His last visit was made in 1929, when he was one of the principal speakers at the inauguration of the Wilmer Institute at the Johns Hopkins Hospital; he subsequently received the Leslie Dana medal at the St. Louis meeting of the National Society for the Prevention of Blindness, and in New York he was made an honorary member of the Academy of Medicine.

A man of great simplicity of character, of great industry, and with a remarkable interest in all subjects, he not only made many valuable contributions to his specialty, but he helped and inspired a host of pupils who are scattered all over the world. His simplicity, kindliness and wideness of interest leave an unforgettable memory.

ARNOLD KNAPP, M.D.

"Death like a mighty stream takes all his sons away."

The world is the poorer by the passing of Professor Fuchs. Ophthalmology deplores the loss of the Master whose life and labors were spread over so many years that it seemed as if he would be with us

ERNST FUCHS, M.D.
1851-1930

always. I desire to pay a tribute of gratitude and respect by recording in a simple way incidents in the late evening of the Master's life, "in his habit as he lived." Old age crept upon him in the environment of his youth in Vienna. There his existence was centered in the university, on the faculty of which his father before him had been a professor.

He long had dwelt in the house that Skoda built, from whose heirs he purchased it. Many will recall those spacious rooms in the Skodagasse, the garden with the large fountain, and the hospitality that was there dispensed. The years brought little change in the immediate surroundings. One would return year after year to find each familiar object exactly as he left it, and the old servants would be there to greet one. Continuously, after the World War, visitors from distant lands were coming and going. Scarcely a day passed in summer without the renewal and knitting together of some old friendship or association. Often members of the younger generation of ophthalmologists, brought up by Fuchs through his textbook, sought interviews that they might say in after years that they had seen the Master. Long they will remember the tall, kindly old gentleman, whose head was bending low, as he showed them something of interest in the house or led them down the spiral stairs into the garden. His was a peaceful, loving home life, marked by all the little courtesies and considerations that go to make up the culture of old Vienna. It was refreshing to see the filial affection exhibited by the son, Prof. Adalbert Fuchs, toward his father. There was the shaking of hands, no matter how often they met and parted during the day. The friends of the father will always be welcomed by the son.

The Master's life was free of excesses of any kind. He never smoked, and he partook sparingly of beer and wine. He went to bed early and rose early. In the evening, he relaxed completely. It was not his habit to work at night; he preferred to get up at 4 o'clock in the morning if necessary. In the palace his contemporary, the Emperor Franz Josef, was said to keep similar hours. Walking was his sole form of exercise. His feats of mountain climbing in earlier years are legendary in Vienna. Latterly he was ever without a hat, and at the end of a season at the country seat at Kritzendorf, his noble head was as brown as a nut. He was a high priest of botany, and but a short time ago he was able to split with his pen-knife a leaf of a box tree into two layers, to demonstrate its structure. During his walks he conversed on many subjects, but seldom on ophthalmology, so entirely could he detach himself from the specialty he adorned. It is sad to contemplate that those hills overlooking the Danube will know the sage of Kritzendorf no more.

He was an accomplished linguist, as was Donders in his time. He spoke and wrote English, French and Italian, and when at 70 he was called to lecture in Spain and South America, he learned Spanish with ease. In his boyhood his father conversed with him in Latin, after the custom of the gentry in Vienna, so that the servants could not understand. His memory was phenomenal. He could carry in his mind an

enormous amount of minutiae. An omnivorous reader, not only of medicine but of such subjects as history, economics, architecture and travel, he seemed to have retained almost everything he ever perused. One marveled at the universality of his knowledge, and one never left him without being stimulated to better efforts.

He had an enormous capacity for work, which continued unabated until the last. It was striking that he who accomplished so much should require so little assistance of any kind. There was no secretary, although the writing and editing of papers constantly went on. Only a few books were at hand. It was evident that the Master was guided by the light that came from within and not from without. His thoughts were set down in shorthand. Usually a paper was written over three times and then sent out to a typist who could read the shorthand. With Arlt, the Master of Fuchs, and the one to whom he most frequently referred, it was different. He wrote slowly and meticulously, so that it was the first draft that went to the publishers. In the preparation of an article from microscopic slides, Professor Fuchs sketched each slide and described it accurately, even though the number might run into the hundreds. Before classifying the information so laboriously gleaned, he would make a schema of the proposed paper, which he faithfully followed. This method he taught to his pupils, insisting above all else that they be careful of the correctness of their statements. To many a young assistant he gave the ideas for his first paper, thus opening the door to him to the domain of literature and production. It was his habit to assign the report of individual cases to the staff, while he himself preferred to make surveys. Writing was with him a diversion and not a task. He no sooner finished one paper than he began another. He had a grasp of the literature and traditions of ophthalmology that no one probably will ever surpass. Having been born in the year of the invention of the ophthalmoscope, his life spanned the entire epoch of modern ophthalmology.

To few men has it been granted to reap the fruits of their labor in so rich a measure. In the leisure of retirement, he studied and classified the vast collection of microscopic preparations of the eye that it took him decades to assemble. At 79 he could do perfect microscopy, sitting three or four hours at a stretch over the microscope without fatigue. So well did he remember what he saw that he could sketch some interesting microscopic field that he had seen days before. There came time and opportunity for travel, a thing he loved to do; he was always longing "strange sights for to see." The Master's peregrinations in all parts of the world as an old man were in the form of visits from the home of one friend to that of another. Honored by universities and learned societies, no word of boastfulness escaped his lips. He lived

to celebrate, among a number of jubilees, the fiftieth anniversary of membership in the German Ophthalmological Society, at Heidelberg, in 1928. Sitting in a high place during a meeting, it was characteristic that at intermissions he was out in the company of more obscure members, for he had much in common with all men.

In late August he paid what was perhaps his last visit to the scenes of his former activities at the Allgemeines Krankenhaus. He went to inspect the library of his old clinic. It was then explained why there were so few books at the home in the Skodagasse. For years he had sent to the clinic library nearly all the books and publications that came to him. "In my own house," said he, "only a few could read them, but here many may profit by them." This incident illustrates one of the Master's salient traits—utter unselfishness. He was always helping others, and yet he was the despair of his family when it came to doing anything for him. Not long after this, while sitting in the garden that Skoda planted, he had a premonition that his days were numbered. Up to this hour he was studying and learning and giving advice with the enthusiasm of youth. When, on November 21, the anticipated summons came, it found the Master prepared and in the complete possession of his unusual mental faculties. Loved, honored, mourned, he has fallen asleep. Immortal is his name.

BERNARD SAMUELS, M.D.

ALLVAR GULLSTRAND, M.D.

1862-1930

The year 1930 will always be remembered with sorrow by ophthalmologists all over the world, on account of the death of three men of international reputation, Theodor Axenfeld, Allvar Gullstrand and Ernst Fuchs.

Allvar Gullstrand stands out, not so much as a clinician, but as a mathematician, who has been engaged in unraveling the laws of the eye as a photographic apparatus.

Being a mathematician of the first rank, and having at his command the Zeiss Works in their entirety, he was able to construct the curvatures in lenses which he had calculated. He originated the punctal lens, the katral lens for aphacia and the aspherical lens for illumination.

His greatest achievement, however, was the slit-lamp, which he discovered and used in the scientific investigation of the posterior curvature of the cornea. With this apparatus, Gullstrand was the first to see the finer structure of the vitreous in the living eye. He lived to see his invention become a part of the necessary armamentarium of every progressive ophthalmologist.

The slit-lamp with the corneal microscope has been the greatest gift which the ophthalmologic world has received in the present generation. It will keep Gullstrand's name by the side of Helmholtz'.

Gullstrand also investigated the mechanism of accommodation, and observed the intracapsular motion of the lens substance during accommodation. It was natural that he was selected to edit the third edition of the first part of Helmholtz' "Physiological Optics," the "bible of the scientific ophthalmologist," as A. von Graefe called it.

The slit-lamp became narrowed to a point, and the demonstration of the pointolite lamp by the inventor will be remembered by many oculists who visited the Washington Congress.

He published his optical investigations in book form in 1911 as "Einführung in die Methode der Dioptrik."

In addition to the mathematical researches, which I am absolutely unable to estimate, Gullstrand also published clinical observations on conical cornea and lenticonus. All this and much more made him the dignified recipient of the Nobel Prize for Medicine in 1911, and his lecture before the commission can be found in the *Archiv für Augen-*

heilkunde (1912, parts 3 and 4, p. 191). He was then professor of ophthalmology in Uppsala, and 49 years old.

He was born in 1862, his father being a well known physician, with a full appreciation of scientific problems. Gullstrand studied ophthalmology under Widmark. In 1911, he presented his reflex-free ophthalmoscope at the Heidelberg meeting wherewith the fundus can be studied in detail, in depth and also stereoscopically. He received his doctor's degree with a thesis on astigmatism in 1888, and became Dozent der Augenheilkunde in Stockholm in 1891. In accepting the professorship of ophthalmology in 1894 at Uppsala, he was able to devote more of his time to scientific research, which he did with well known results.

To quote J. W. Nordenson, "His profound differential geometric studies of the laws of the focusing of the rays of light and of image formation make a monumental work, which places the author among the great scientists who have contributed to the progress of our knowledge of optics. They bear witness to an extraordinary mathematical imagination whereby he was able to see geometrically (with his mind's eye), the most intricate analytic geometric methods of computation."

After 1912, Gullstrand was for many years a member of the Council of the Heidelberg Ophthalmological Society, and received in 1928 the von Graefe medal, which is given every ten years "to the one among the contemporaries, without distinction of nationality, who has done most for the progress of ophthalmology." This medal was given in 1886, for the first time, to Helmholtz, later to Theodor Leber, Ewald Hering and Carl von Hess. Gullstrand was recognized as "the genial mathematician and physicist among ophthalmologists, to whose thoughts and investigations we owe new methods and instruments, which have produced unexpected progress in all directions."

Interest in his teachings is continued in his country, as contributions to the *Acta ophthalmologica* prove.

Many of us will remember Gullstrand's striking personality at the Washington Congress in 1922. He will be remembered with deep appreciation for his extraordinary talents.

E. E. Blaauw, M.D.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

PIGMENTED CELLS IN THE SCLERA OF VERTEBRATES. E. PANICO, Ann. di ottal. e clin. ocul. **58:** 338 (April) 1930.

Previous descriptions of these cells, which occur only in certain animals, are reviewed. The author examined forty types of vertebrae, including man, for the presence of such cells. In man they are found especially around the entrance of the optic nerve. Where the scleral fibers split to form the dural sheath and lamina cribrosa the cells are irregular, while further from the nerve they are more fusiform. Along the course of the sclera a few such cells are found, chiefly in the layers nearest the choroid, and sometimes they are more numerous about the perforating vessels. A few are present near the limbus. In the eyes of Negroes the distribution is the same, but the cells are more abundant.

In the ox and horse an abundance of such cells is found throughout the sclera. In other animals, including birds and fishes, the distribution varied greatly, the cells being abundant in birds and mammals, scanty in reptiles and amphibia and scanty or absent in fishes. The cells showed a definite relation to the amount of pigment present elsewhere in the eye, being absent in white mice, white rabbits and white doves. The cells are homologous to the chromatophores of the choroid and are morphologically identical with these. The pigment granules in both types of tissue are round or slightly elongated. In the horse, dog and sheep, pigmented cells are to be found in the lamina cribrosa.

S. R. GIFFORD.

DEVELOPMENT AND MORPHOLOGY OF THE CANAL OF SCHLEMM. R. SONDERMANN, Arch. f. Ophth. **124:** 521 (July) 1930.

The primordium of the canal of Schlemm is a system of radial vessels which during the first and the second half of the third month carry the blood from the tissues in front of the margin of the eye cup toward the ciliary veins. A primary circular vessel cannot be seen. The radial vessels go along the inner surface of the sclera and finally enter the sclera. This part of the vessel becomes (apparently because of increased pressure) ectatic, while the rest of the vessel obliterates. The ectatic parts communicate with each other by ectatic capillaries, and thus the canal of Schlemm is formed. This mode of development explains why in the adult eye serial sections of the region of the canal of Schlemm show alternatingly one large lumen and several small "plexus"-like lumina. The spaces of the pectinate ligament communicate with the canal of Schlemm by means of small canaliculi (inner canaliculi) which usually empty into the canal near its posterior end. The canaliculi which communicate with the anterior ciliary veins (outer canaliculi) leave the canal near its anterior end.

P. C. KRONFELD.

Blind

CAUSES OF BLINDNESS AMONG CHINESE. S. P. CHANG, Nat. M. J., China **16:** 370 (Aug.) 1930.

From May, 1928, to April, 1929, 336 (10.2 per cent) blind patients were seen among 3,279 patients admitted to the eye clinic of the Peiping Union Medical College Hospital. In 18.9 per cent of these blind persons, the cause of blindness could not be ascertained. The main determinable causes were in the following descending order of frequency: keratomalacia, 34.5 per cent; trachoma, 14 per cent; glaucoma, 8.9 per cent; syphilis, 5 per cent; cataract, 5 per cent; gonorrheal ophthalmia, 5 per cent; injuries, 4.2 per cent; tuberculosis, 2.1 per cent; neoplasms, 0.6 per cent; smallpox, 0.3 per cent. With the exception of keratomalacia and an interchange of place of glaucoma and cataract, the main causes of blindness occupied the same relative position in Ling's series and in Chang's analysis of cases. Keratomalacia was an insignificant cause, 0.52 per cent (in only four eyes) in Ling's 760 blind patients, while it was responsible for 116 of Chang's series of 336 blind, or 34.5 per cent. The cause of this remarkable difference is chiefly the civil war, as during the last year a great number of the patients with ocular conditions were soldiers. On account of the restricted finances of the armies and the difficulty of seeking aid in the early stage of the disease, keratomalacia spread. [J. A. M. A.]

Congenital Anomalies

A CASE OF FAMILIAL CONGENITAL ANIRIDIA. Y. I. ULIANITSKY, Russ. Opht. J. **11:** 671 (May) 1930.

The author reports a case of aniridia. This condition was observed in five generations. The patient's father, grandfather, son, two sisters and his sister's children had "black eyes." With the exception of a very narrow border, 0.5 mm. wide, above and temporally, there was an absence of the iris in both eyes of the patient. All the members, except those of the last generation, had marked opacities of the anterior capsule of the lens. All the lenses were subluxated upward, nasally or temporally. None of the persons had nystagmus, and all had normal corneas, both in size and transparency.

The author shows by diagram that the transmission of aniridia from generation to generation has no regularity. O. SITCHEVSKA.

Cornea and Sclera

PIGMENTED NETWORK ON THE POSTERIOR SURFACE OF THE CORNEA. Y. SHOJI, Arch. d'opht. **47:** 129 (March) 1930.

After describing a case in which he had found a patch of pigmentation in the form of a network on the posterior surface of the cornea, the author lists sixteen conditions which may produce pigmented changes in the cornea and anterior chamber. The literature of this subject is discussed, and three modes of development are cited. They are: (1) acquired inflammation or perforation of the cornea with adherence of

the iris to its posterior surface; (2) congenital adherence due to an intra-uterine inflammation, and (3) an embryonic malformation.

The first two are more commonly thought to be the explanation. The author feels that the third hypothesis best explains his case. The embryologic development of the pupillary membrane is discussed in association with the formation of the anterior chamber.

S. B. MARLOW.

VACCINIAL KERATITIS. P. TOULANT, Arch. d'opht. **47**: 229 (April) 1930.

After referring to the literature of this subject, the author reports a personal case. Two clinical forms have been described. The first is benign and consists of infiltration of the margin of the cornea, which rapidly becomes vascularized and disappears without leaving a trace. The more common form is the vaccinial pustule, which is much more serious. It is the result of direct inoculation. It begins with an ulcer; no vesicle has ever been described. The incubation period varies from twenty-four hours to five days. The disease progresses in spite of treatment, its course being prolonged and painful. The particular receptivity of the eye has been extensively studied. Although vaccination may produce a general immunity, the cornea does not share in this. However, a cornea once inoculated cannot be reinoculated. Treatment is entirely symptomatic once the disease is established. Prophylaxis should be emphasized because of the seriousness of ocular inoculation. The physician should wear protecting glasses, and he should have at hand 1 per cent silver nitrate, or 10 per cent strong silver protein, or 2 per cent chloramine—an antiseptic regimen such as is used in the prophylaxis for ophthalmia neonatorum.

S. B. MARLOW.

TWO ATYPICAL CASES OF KERATITIS NODOSA GROENOUW, WITH HISTOLOGIC EXAMINATION. P. M. LADEKARL, Acta ophth. **8**: 213, 1930.

A meticulous review is made of corneal dystrophies which are characterized by a degenerative process occurring either in the form of circumscript patches, nodules or lines in the superficial layers of the cornea. The author reports two cases which he believes should be included in this group because of similar clinical pictures and histologic signs. In this classification, Ladekarl includes the reticular keratitis described by Biber, Haab and Dimmer and the patchy familial corneal degeneration described by Fehr, which differs from Groenouw's description merely in that it is less superficial. They have in common a chronic progressive course, a pathologic process which is localized in the superficial layers of the cornea and a familial and hereditary incidence. The designations, patchy, nodular and reticular, are only an expression of the varying arrangement of the opacities in diseases of the same nature. The various types have been observed in the same family. Most authors agree as to the degenerative nature of the condition, but opinions differ with regard to the primary cause. Groenouw thinks that the disease arises as a sequel to nutritive disturbances based on a hereditary basis,

and Fuchs attributes the nutritive disturbance to endocrine dysfunction; whereas Pillat believes that there is a degeneration of the trigeminus which produces the lesion.

The author found that in excision of the opacities the condition improved. Others, however, record unfavorable results from this therapeutic measure.

A. M. YUDKIN.

Experimental Pathology

ON THE PROPERTIES OF THE EXTERNAL OCULAR COATS AT ABNORMAL INTRA-OCULAR PRESSURES. F. P. FISCHER, Arch. f. Augenh. 103: 1 (June) 1930.

These experiments were undertaken to determine the physical properties of the ocular coats in eyes with hypertension. For this purpose, an investigation was made of the water content of the ocular structures as follows: the capacity, i.e., the extent to which the water may be bound, and the intensity, i.e., the tendency to hold water, and the form of the water content. The original article must be read for a description of the methods employed.

Preliminary experiments on the corneas and scleras of rabbits showed the following: The water content of the cornea is greater, and the salt content less than the sclera. The relation of the ash of the cornea to that of the sclera is 3:2. The sclera contains more organic material than the cornea.

The cornea swells in water, but not the sclera. Both tissues swell in acids, but the sclera to a greater degree. In strong acids, however, the swelling decreases with increasing concentration.

In alkalis, the cornea swells more than the sclera and more than it does in water. In equally dissociated acids and alkalis, the cornea and sclera swell equally, but the swelling is greater in acids than in alkalis. Salts inhibit the swelling, both that due to acids and alkalis, but particularly the former. Nondissociated crystalloids exert different influences on the degree of swelling.

The cornea swells more rapidly than the sclera. Both tissues swell in a radial direction due to the regular arrangement of anisodiametric micellae, which form the histologic fibers.

The specific gravity of the cornea at its normal water content is less than the sclera dehydrated to the same degree. While the specific gravity of both increases equally up to a 60 per cent loss of water, both suddenly increase beyond this level, the cornea steeper than the sclera, so that when dried to a constant weight the specific gravity of the cornea is much greater than that of the sclera.

The sclera gives off water much more quickly than the cornea. In sixty minutes, the sclera has reached a constant weight, whereas the cornea takes eighty minutes (temperature, 37 C.).

The refractive index of the cornea is greater than that of the sclera, from 1.378-1.379 to 1.375-1.376, and increases to a greater extent with increasing dehydration.

The intensity with which water is bound is greater for the cornea than for the sclera, and the form of the water-binding is different in the two tissues.

In the main part of the investigation, fifty-one human eyes were examined, twenty-six without and twenty-five with rise of intra-ocular pressure. In both groups, the water content of the corneas was identical or only slightly different in every respect. That of the sclera, however, was different in the two groups. The water content was less in the eyes with raised tension. The salt content of the sclera in these eyes was greater, and there was more ash. These scleras reached a greater degree of swelling in water, acids, alkalis and nonconducting crystalloids. In organic acids and in salt solutions, the swelling was inhibited to a greater degree. The rapidity with which these scleras can be dehydrated is greater than that of those with normal or lowered tension.

The intensity of the water-binding is greater also. The cause of these changes is seen in the different arrangement of the colloids. These changes, however, are not due directly to the increased pressure, nor is this caused by the changed physical properties of the sclera, but both come from a common complex cause, which is not local in the eye itself, but affects the whole organism.

F. H. ADLER.

XEROPHTHALMIA IN RATS AND "PERIOCULAR REACTION." S. V. GUDJONSSON, Acta ophth. **8:** 184, 1930.

A splendid and instructive group of photographs of the ocular changes observed in albino rats fed on a diet deficient in vitamin A is offered by Gudjonsson. He has chosen the important stages of the disease to depict the destruction which is produced by this diet. The photographs clearly illustrate the clinical description which the abstractor described in *The Journal of the American Medical Association* (**79:** 2206 [Dec. 30] 1922).

A. M. YUDKIN.

General Disease

A CASE OF MIKULICZ'S DISEASE. H. B. OWEN and R. S. F. HENNESSEY, Brit. J. Ophth. **14:** 582 (Nov.) 1930.

An African, aged between 50 and 60, had enormous swellings in both orbits and bulging of the lids, greater in the upper lids. On the right side was a firm movable mass occupying the upper region of the orbit in front of the eyeball, evidently a greatly enlarged lacrimal gland. Below this was a similar smaller growth springing from the palpebral conjunctiva. Occupying the lower region of the orbit was a mass of moderate size, originating in the palpebral conjunctiva of the lower lid. On the left side was a similar condition, but the lacrimal gland showed greater enlargement, and the palpebral conjunctiva of the upper lid was the seat of a much more profuse growth with edema of the overlying skin and ulceration of the conjunctival portion of the margin of the lid. In the lower region of the orbit was a comparatively small mass again taking its origin from the palpebral conjunctiva. In the left eye, the bulbar conjunctiva showed extensive general invasion by growth, while in the right it was but slightly affected near the fornices; ocular movements were limited for this reason, the limitation being slight on the right side and marked on the left. A divergent squint was present on the left side, and was presumably due to the same mechanical cause.

Except that senile lenticular opacities were present, the internal structures of the eye were normal. While the parotid glands were not enlarged, the submaxillary and sublingual salivary glands showed considerable firm, nodular enlargement. There were a number of small, firm, nodular swellings in the lips and cheeks adherent to the mucous membrane and derived from the buccal glands. The hard palate showed a similar condition, though the swellings were smoother and smaller. The tongue was not affected. The cervical and axillary glands were markedly enlarged, being firm, discrete and movable. Evidence of syphilis were absent, and the Sachs-Georgi test was negative.

Operative treatment was obviously indicated. The enlarged lacrimal gland and the lower orbital masses were removed under general anesthesia.

The patient was the subject of ankylostomiasis, but the elimination of ankylostomes produced no appreciable effect, either on the general health or the red cell count. The tumors consisted of a dense mass of lymphoid cells with an occasional cell of endothelial type, the whole embedded in a fine stroma. The tissue was poorly supplied by blood vessels. Neither giant cells nor eosinophils were present. No tendency toward fibrosis was present. In the lymph gland, the original fibrous trabeculae had practically disappeared. No distinction could be discovered between cells causing the enlargement of the lymph gland and the original lymphoid cells.

The authors consider it difficult to classify this case under any of the headings proposed by von Brunn, of cases of Mikulicz's disease presenting alterations in the blood, into those with severe anemia, lymphatic pseudoleukemia and aplasia of the bone marrow and those with a frank leukemia. Time alone can show whether the case described is to be placed among the leukemias or is to prove one of a group differing markedly from those cases of Mikulicz's disease which have hitherto been reported.

W. ZENTMAYER.

Glaucoma

GLAUCOMA. G. CIRINCIONE, Ann. di ottal. e clin. ocul. **58:** 291 (April) 1930.

This is a continuation of the posthumous notes of the author. In this section he discusses the various operations for glaucoma and their supposed mechanism. He believes iridectomy is effective by establishing better drainage between the anterior and posterior chambers since his idea of the primary cause of glaucoma is not closure of the chamber angle but increase in the volume of the vitreous. Iridectomy is insufficient in advanced cases of chronic glaucoma, and here he preferred corneoscleral trephining with complete iridectomy, although according to Lagrange, sclerecto-iridectomy is described as giving equally good results.

He distinguishes a special type of posterior glaucoma, with no pain, slight rise of tension at times and marked cupping of the disk, which he believes is caused by loss of the elasticity of the sclera which exerts increased pressure on the vitreous. He finds operation to be of no effect in this type, and thinks miotics are the only form of treatment indicated.

In hydrophthalmos, also, he considered that operative procedures are usually disastrous, and here miotics are without effect. Anteriosclerotomy is the only procedure which may, in some cases, be attempted.

S. R. GIFFORD.

Lacrimal Apparatus

CONTINUED INVESTIGATION ON THE LACRIMAL PASSAGES. K. G. PLOMAN, Acta ophth. **8:** 155, 1930.

By means of roentgenographic pictures and volumetric studies, Ploman finds that there is a regular compression and dilatation of the lacrimal sac and that the compression of the sac is more pronounced in contraction of the eyelids. Under normal conditions when the lids reopen in winking, the pressure of the orbital tissue on the lacrimal sac is diminished, its walls become placid, and in those cases in which there has been actual compression, the sac resumes its former volume. Suction is exerted on the liquid in the lacus by this process. Under physiologic conditions, when the lids are kept open there is probably no hindrance to the suction of the liquid from the conjunctiva to the lacrimal sac. The surface tension at the nasal opening of the canal prevents liquid from being drawn up from the nose. This would disturb the pumping mechanism by the introduction of backwater. The canaliculi represent the most important part of the pumping mechanism, and the sac plays a minor part in the conveyance of tears into the nose.

A. M. YUDKIN.

Lens

ELECTRIC CATARACT. T. B. HOLLOWAY, Am. J. Ophth. **13:** 595 (July) 1930.

The author reports the case of a boy, aged 14, whose vision became impaired twenty-three months after an electric shock, and whose lenses, six months later, were completely cataractous. Laboratory tests and physical examination gave negative results. Both eyes were operated on successfully.

W. S. REESE.

THE PATHOLOGY OF HUMAN CATARACT. PART I. THE WEIGHT RELATIONS OF HUMAN LENSES. J. KUBIK, Arch. f. Augenh. **102:** 657 (Feb.) 1930.

The author reviews the work done by previous investigators on the changes in weight of human lenses, and points out that aside from the work of Priestley Smith all other authors have dealt with too limited material to draw conclusions of value. In this present paper the weights of 433 lenses which were extracted in the Elschnig clinic by the intracapsular method are given. Table 1 shows the relation between weight of the lens and the age of the patient.

It is quite apparent, therefore, that the weight of the lens increases with age. When the lenses were grouped according to the refractive condition of the eye from which they were taken, it was seen that with

the increasing size of the eye the lens increased in weight. In other words, the lenses from hypermetropic eyes weighed less than those from myopic eyes. Table 2 shows the relationship between the weight of the lens and the character of the cataractous change.

TABLE 1.—*Relation Between Weight of Lens and Age of Patient*

Age	Average Weight, Mg.	Number of Cases
45	166	24
50	176	22
55	190	27
60	194	59
65	204	76
70	207	92
75	204	82
80	204	34
85	218	14

TABLE 2.—*Relation of Weight of Lens and Cataractous Change*

Form of Cataract	Average Weight, Mg.	Average Age	Number of Cases
Incipient	206	66.7	29
Cortical, anterior and posterior	179	64.5	59
Praec. nucl.	220	66.1	62
Almost mature	209	66.6	138
Mature	188	64.7	58
Hypermature	174	69.2	18

With the formation of cataract, there is a gradual diminution in the weight of the lens, and the more ripe the cataract the greater is this loss of weight.

A discussion follows of the weights of various forms of cataract in which there were too few cases to draw any positive conclusions.

F. H. ADLER.

LEAF-SHAPED OR ROSETTE-SHAPED CATARACTS. W. RAUH, Klin. Monatsbl. f. Augenh. **84:** 766 (June) 1930.

This type of cortical cataract was first described by Vogt, who judges them to be of traumatic origin. The opacity, first directly below the capsule, moves toward the nucleus by apposition of new and clear fibers. Handmann doubts the traumatic origin in similar cases of his own. Now Rauh reports seven new cases. In every one the rosettes were located in the cortex and were limited to one single layer. The fundus was normal in all but one case; vision was reduced in five cases, and an injury could be traced in four of them. One of the rosettes was connected with the capsule by two strands crossing the clear cortex.

Of the twenty-two cases published by Rauh and the two authors mentioned, nineteen occurred in men. In no case were both eyes affected. These facts, in the author's opinion, are indicative of a traumatic origin. Reexaminations, during a period of years, may allow measurements of the gradual motion of the opacities toward the nucleus.

K. L. STOLL.

THE LENS CAPSULE IN INTRACAPSULAR EXTRACTION OF CATARACT. A. RÔTTH AND N. KLEIN, Klin. Monatsbl. f. Augenh. **84:** 823 (June) 1930.

Extraction of the cataract in the capsule by means of forceps is the method producing the least reaction. It is essential for success that the capsule be stronger than the zonula, a condition which cannot be recognized in advance. The authors made microscopic examinations of sixty-nine lenses extracted with Kalt's forceps at Joseph Imre's University Eye Clinic at Pecs, Hungary. They found the anterior capsule thinnest at the equator (it was thinnest at the anterior pole in normal lenses examined by Salzmann), and the average for the posterior capsules lower than the average found in normal lenses. The anterior capsules were thicker than in normal eyes, the lowest range being the same in both types at the same age. This means that they are favorable for extraction with forceps, and that the thickness of the capsule is not of primary importance for success. The tightness of the capsule, greatest in swollen and hard cataracts, and its elasticity are of major importance.

K. L. STOLL.

BLEPHAROCHALASIS OF THE LOWER LIDS. R. STEIN, Klin. Monatsbl. f. Augenh. **84:** 846 (June) 1930.

Two typical cases of blepharochalasis with atrophy of the epidermis, weak fascia, ectasia of the subcutaneous blood vessels and displaced lacrimal gland are described. The lower lids were involved, a condition hitherto not described. In one case the levator, and in some measure the superior rectus were involved. Dermatolysis had weakened the connecting fibers between the tendon of the levator and the inner surface of the skin, causing ptosis. In the second patient, the lid of one eye was greatly atrophied and extended. No new information was obtained regarding the etiology.

K. L. STOLL.

INJECTIONS OF ALCOHOL IN SPASTIC ENTROPION. O. A. DUDINOFF, Russ. Opht. J. **11:** 809 (June) 1930.

The author treated ten patients with spastic entropion. Most of the patients were elderly people. Five of them received 80 per cent alcohol, and the other five alcohol mixed with a 2:1,000 dilution of iodine. The following technic was used: One cubic centimeter of 2 per cent procaine hydrochloride is injected under the skin 3 mm. from the margin of the lid near the external angle. In five minutes, 80 per cent alcohol or iodinized alcohol is introduced at the same place. The lid becomes edematous, the edema spreading toward the cheek and temple in about twenty-four hours. This gradually disappears in from eight to ten days. The injections were painless.

An immediate correction of the entropion resulted in eight patients, three of whom suffered from complete entropion, and five from partial trichiasis. Two patients had deep conjunctival scars of trachomatous origin. It was necessary to perform an operation in one case in addition to the injection of alcohol. In the second patient, an injection of alcohol was given to alleviate the discomfort following an unsuccessful operation.

The virtue of the injections, according to the author's opinion, is due to the combined action of the paralysis of the nerve endings with the formation of firm connective tissue in the orbicularis muscle.

The author's conclusions are:

1. Injection of alcohol in spastic entropion is a safe and simple procedure which gives very good results.

2. It is indicated in entropion caused by ocular operations, and in corneal involvements caused by entropion.

3. Injection of alcohol is used as an auxiliary factor to an operation not sufficient to correct the entropion.

4. Since the technic of the injection is simple, it is superior to operative measures, wherever the result of the operation does not guarantee against the recurrence of the entropion. O. SITCHEVSKA.

Neurology

THE IMPORTANCE OF CHOKED DISK IN THE DIAGNOSIS BETWEEN EPIDEMIC ENCEPHALITIS AND CEREBRAL TUMOR. G. WORMS and G. SOURDILLE, Arch. d'opht. **47**: 193 (April) 1930.

The authors report in detail a case observed for six years in which a diagnosis of encephalitis was made, until bilateral choked disks appeared, which led to complete surgical cure. They emphasize the relationships between ophthalmology and neurology. Encephalitis can resemble almost all neurologic syndromes, and mistakes will arise for this reason, especially during an epidemic. In this condition, however, choked disk is rare, and when it does occur it develops rapidly, recedes spontaneously and recurs. The authors agree with the conclusions presented by Knud Winther and Fribourg-Blanc. They feel that in the presence of choked disk the diagnosis of encephalitis can be allowed to stand only after every other possible cause has been eliminated by the most exhaustive study. S. B. MARLOW.

ANATOMIC AND CLINICAL PROOF OF THE MECHANISM OF THE HYPOPHYSIS IN OPHTHALMIC MIGRAINE AND OPHTHALMOPLEGIC MIGRAINE. J. SÉDILLOT, Arch. d'opht. **47**: 209 (April) 1930.

The author first refers to the recurrent discussions on the mechanism producing migraine, which have never led to any satisfactory conclusions. The most recent is the thesis of Cantilo published in 1929. The scintillating scotoma is an irritative phenomenon of a nervous conductor, and the hemianopia an inhibitory phenomenon of the same nervous conductor. In his opinion, they are both due to the sudden compression of the optic tract by the hypophysis when it is pathologically swollen in an erectile process.

The anatomic location of the hypophysis in the sella turcica, the anterior and posterior walls of which are bony and therefore resist swelling, necessitates expansion laterally where the walls are fibrous. The optic tracts are in close relation at these points and can thus be subjected to pressure. In the first stages of this sudden compression, the irritative phenomena arise, later the hemianopia. In his argument against the possibility of the retina being the site of the lesion, the author recalls the famous observations of the physician Plateau, who

had the typical scintillating scotoma in spite of the fact that he was blind from an extensive choroiditis.

Further evidence as to the site of the lesion being in this region is the dilation of the pupil of the eye on the side of the hemianopia. All other symptoms can also be explained by the author's theory. The olfactory nerve can be compressed. The motor aphasias and loss of speech are explained by compression of the anterior communicating artery. If spasm or constriction of the posterior communicating artery is accepted as the cause, then there will be considerable difficulty in explaining the cortical conditions. He suggests that ophthalmoplegic migraine is a paralysis of the third nerve often due to syphilis, which has developed earlier than it would have if migraine were not present.

S. B. MARLOW.

TWO CASES OF HOMONYMOUS HEMIANOPIA AND HEMIANOPIC QUADRANT DEFECT OF CORTICAL ORIGIN. V. BARLETTA, Ann. di ottal. e clin. ocul. **58:** 353 (April) 1930.

One of the author's cases was of complete right hemianopia with sparing of the fixation point, due to cerebral thrombosis. The other was of a defect in the left lower quadrant with evidently some involvement of the fixation point, since vision was 7/10 in the right eye and 2/10 in the left. The lesion was the result of syphilitic endarteritis. Examination for the Wernicke reaction was unsatisfactory in both cases. In an attempt to localize the lesions, the author reviews the vascular supply of the various portions of the visual pathways, and the associated cerebral symptoms following occlusion of the various large vessels concerned. The optic tract is supplied by the anterior choroidal and posterior communicating arteries; the optic radiation by the anterior choroidal, sylvian and posterior cerebral arteries, and the cortical centers by the posterior cerebral artery. Complete occlusion of the posterior cerebral artery gives rise to the thalamic syndrome of hemianesthesia, hemiakinesia, painful phenomena, disturbances of coordination and athetosic movements besides a more or less extensive hemianopia, and in the case of occlusion of the left posterior cerebral artery also to sensory aphasia and alexia. The thalamic symptoms may be minimal or absent in partial occlusion. Closure of the sylvian artery leads to death, and only lesions of the subcortical branches are of interest here as causing hemianopia. If the artery is involved before the common trunk of the ascending arteries is given off, hemiplegia and sensory disturbances will result, in addition to hemianopia sparing the macular area. A left-sided lesion of this type also causes motor aphasia. Occlusion beyond this point involves the posterior superficial branches of the sylvian artery and, if left-sided, produces motor aphasia and motor apraxia as well as hemianopia. If right-sided, only the hemianopia will be noted.

Lesions of the anterior choroidal artery involve the posterior part of the internal capsule and may produce hemiplegia or hemianesthesia as well as hemianopia, so that it can only be distinguished from a sylvian lesion if left-sided, as aphasia is absent. In the author's first case, he believes that lesions of the left anterior choroidal and sylvian arteries can be excluded as no other cerebral symptoms were present, and that hence the lesion of the left posterior cerebral artery must be held responsible, and that the lesion must have been a small one, practically

limited to the cuneus. In the second case, a right-sided lesion was present, and hence a lesion of the sylvian cannot be excluded by the absence of aphasia. Thus the choice would lie between a partial lesion of this artery and one of the posterior cerebral artery. This the author thinks cannot be decided more exactly, although according to Schiff-Wertheimer, a defect of the inferior quadrant such as was present here is nearly always due to a sylvian lesion, while a defect of the upper quadrant would indicate a lesion of the posterior cerebral artery.

S. R. GIFFORD.

Ocular Muscles

FUNCTIONAL SYNERGY OF MASTICATION AND ELEVATION OF UPPER EYELID. M. MARIN AMAT, Siglo méd. **86:** 57 (July 19) 1930.

Marin Amat reports the case of a child, aged 8, whose parents (in whom there was no consanguinity) and six brothers and sisters were normal. The general mental and physical condition of the child was normal, except for a typical synergy of mastication and elevation of the right upper eyelid. The abnormality was congenital, unilateral and accompanied by total ptosis of the eyelid. The eye opened only while the child either opened his mouth or moved his lower jaw to the left. When the eye was opened, a paralytic convergent strabismus (caused by paralysis of the external rectus) was observed. The visual acuity was normal in both eyes. The pathogenesis and treatment of this condition are unknown. An origin similar to that of other synergies (in which a functional association of the cerebral nerves has been held responsible for the condition) is suspected in these cases. The author reviews the literature and makes reference to a personal observation (previously reported) in which the inverse phenomenon existed (the eye was constantly open and closed only when the mouth was opened). Two hypotheses are given to explain the phenomenon: (1) the intervention of motor fibers arising from the masticator nerve (in the motor portion of the trigeminus), by exercising an abnormal influence on the levator palpebrae superioris muscle, and (2) the presence of an anastomosis between the nuclei of origin of both cerebral nerves between which the functional synergy exists. The only scientific proof is the electrical exploration of the oculomotor nerve in its entire course. The most plausible explanation of the case under discussion seems to be that the functional synergy between the levator palpebrae superioris muscle (innervated by the oculomotor nerve) and the depressor muscle of the inferior maxilla (innervated by the masticator nerve) is caused by the presence of an abnormal peripheral anastomosis between the oculomotor and the masticator nerves. [J. A. M. A.]

Orbit, Eyeball and Accessory Sinuses

THE NOSE AND THROAT IN RELATION TO ORBITAL AND OCULAR DISEASE. J. LEWIN, Brit. M. J. **2:** 820 (Nov. 15) 1930.

The author believes that the most probable path for an extension of an infection from the ethmoid labyrinth to the orbit is along the line of the ethmoid vessels. One reason for this is found in the observation

that pus occurring in the orbit in orbital cellulitis is always found in the upper and inner part of this cavity. Then the passage of infection by this route accounts for two of the most common and important complications, namely, cavernous sinus thrombosis and meningitis. Thrombosis is carried along the radicles and trunks of the ethmoidal vein which opens into the ophthalmic vein and so into the cavernous sinus. As the ethmoidal vessels run a part of their course within the cranial cavity under the dura, an entry for infection of the meninges is thus given. This location of the infection also explains that orbital cellulitis should be treated by a radical operation on the nasal sinuses by the external route.

A second condition discussed is the enlargement of the blind spot. The author believes that the relationship of the blind spot to primary nasal infection is but little appreciated. He thinks that this enlargement is explained by a blurring and a loss of visual acuity in that part of the retina immediately surrounding the optic disk. It is, in fact, an inflammatory lesion of these retinal fibers. This, the van der Hoeve sign, is an important symptom of diseases of the posterior sinus. He quotes one observer who in 102 cases of ethmoidal and sphenoidal diseases found the blind spot enlarged in 31 per cent. In the author's opinion, the antrum is as frequently at fault, and he quotes a case in illustration.

He believes that this ocular element is not an infection, but rather compression by inflammatory exudate at the back of the orbit, which belief, he thinks, is supported by the rapid improvement after treatment of the primary nasal trouble.

Finally, he speaks of phlyctenular conjunctivitis and blepharitis which he finds to be secondary most frequently to a focus in the tonsils; the results of tonsillectomy are so good that this operation should be undertaken even in the absence of any apparent tonsillar disease.

A. KNAPP.

Refraction and Accommodation

CONTINUED CRITICISM OF THE ORIGIN OF MYOPIA. G. LEVINSOHN, Arch. f. Augenh. **102**: 308 (Dec.) 1929.

The author defends his theory of the genesis of myopia against the criticism of Scheerer and Buckler. He maintains that the senile changes are essentially different from the pathologic changes seen in myopia.

F. H. ADLER.

Retina and Optic Nerve

LIPEMIA RETINALIS. W. R. PARKER and A. M. CULLER, Am. J. Ophth. **13**: 573 (July) 1930.

Two carefully studied cases are reported. The first was that of a young farmer who developed acidosis and lipemia retinalis. Total blood fat was 11.6 per cent. Under treatment, the acidosis and lipemia retinalis disappeared, at which time the blood showed 2.26 per cent fat. He was then placed on a high fat, high caloric diet. No changes in the fundus occurred, thus controverting the theory that lipemia retinalis is produced by fat in the diet. The blood fats did not rise during this

experiment. When acidosis was allowed to develop, the lipemia retinalis reappeared when the blood fat was 3.65 per cent. The second case was that of a high school student who had acidosis and a blood fat of 4.28 per cent. Under treatment, the lipemia retinalis disappeared when the blood lipids fell to 2.83 per cent. Thirty-seven reported cases are summarized as to sex, age, level of blood lipids, etiology, associated diagnosis, prognosis and ophthalmoscopic aspect. The following summary is given:

1. Apparently no disease except diabetes uncomplicated by treatment produces a lipemia of sufficient degree to be recognized ophthalmoscopically.

2. Diabetic persons probably do not show lipemia retinalis unless they have acidosis.

3. Lipemia retinalis is confined largely to younger diabetic patients because they have a less efficient fat metabolism than older people.

4. Roughly, lipemia retinalis occurs when blood fats rise above 3.5 per cent and disappears when blood fats fall below 2.5 per cent.

5. Lipemia retinalis does not alter the prognosis in cases of diabetes.

W. S. REESE.

BLOOD CALCIUM IN DIABETIC RETINITIS. R. D. LAWRENCE, K. MADDERS and H. R. MILLAR, Brit. M. J. **2**: 559 (Oct. 4) 1930.

Ten unselected patients with retinitis were examined by Lawrence and his associates. They were unable to detect any abnormality in the serum calcium contents of these patients. The average serum calcium in these ten cases was 9.6 mg. per hundred cubic centimeters; the lowest figure was 8.6, and the highest 11 mg. In several cases, the amounts fell between 9 and 10.5 mg., and in no case, therefore, was an abnormally low or high figure obtained. Five of the patients were men and five women. The average age was 61 years, the youngest being 44 and the oldest 71. Seven cases were severe enough to require insulin. The minimum ascertainable duration of the diabetes was two years, the maximum twenty years. Three patients showed traces of albuminuria without other evidence of Bright's disease; in them, the retinal picture was that of diabetic retinitis alone. Gross arteriosclerosis was not a noticeable feature. The mean systolic pressure was 150 mm. of mercury, the diastolic 90; the highest systolic pressure was 185, the lowest 100. It is clear, therefore, that true Bright's disease and hyperpietic arteriosclerosis were not important etiologic factors in these cases, which appear to be really "diabetic" in nature. [J. A. M. A.]

THE CLOSURE OF TEARS IN THE RETINA. R. RUBBRECHT, Arch. d'opht. **47**: 160 (March) 1930.

This paper is a description of the operative procedure used by the author in localizing and cauterizing retinal tears. After determining the meridian in which the tear is found, the limbus is scratched on both sides of the cornea with a discission needle dipped in india ink. A knotted black thread is then inserted on one side and left loose. The distance from the limbus is then determined by adding 8 mm. to the

number of disk diameters that the tear is situated from the most anterior visible portion of the retina. The sclera is next laid bare, and the loose end of the knotted thread carried over the cornea in line with the second scratch in the limbus. A line is then drawn on the sclera along the thread with a knife dipped in india ink. The sclera is then incised at the proper distance from the limbus, the length of the incision being from 4 to 5 mm. Care is taken not to penetrate too deeply through the sclera and thus produce new holes in the retina. The whole length of the incision is then cauterized with an electrocautery. The author's reason for such a long incision and cauterization is the possibility of error in exact localization and the possibility of catching both sides of a wide tear in the resulting scar. He is careful not to penetrate deeply into the vitreous with his cautery.

S. B. MARLOW.

RETINAL DETACHMENT. A. ELSCHNIG, Arch. f. Augenh. **103:** 271 (June) 1930.

It is generally considered that a retinal detachment is cured when the retina becomes reattached. Elschnig points out that this is incorrect for two reasons. It is only in exceptional cases that the vision returns after a complete reattachment occurs. In many cases, in spite of a reattachment, there is further loss of vision, and there are cases in which the detachment remains but is well demarcated and the visual acuity improves.

In this paper, he reports a number of cases demonstrating these points. He draws particular attention to those cases in which a retinal detachment in the periphery stays the same from year to year without anything being done to the eye. He feels that too little attention has been paid to this type of case. He acknowledges that the Gonin operation is a definite forward step in the treatment of retinal detachment, but he is puzzled as to the manner in which this improvement occurs. He points out that all the older ophthalmologists directed their efforts in curing detachment of the retina to creating a hole in the retina so as to allow the fluid to escape into the vitreous, whereas Gonin attempts to cure retinal detachment by the closure of a perforation already existing.

He reports eight cases of detachment in which the Gonin method was employed in his clinic.

F. H. ADLER.

INCONGRUITIES BETWEEN CHOKED DISK AND THE CAUSATIVE CONDITION. H. MEYER, Klin. Monatsbl. f. Augenh. **84:** 671 (May) 1930.

The intensity of the choked disk in intracranial processes ordinarily depends on the intensity of the intracranial pressure. This may be observed in spontaneous fluctuations in the causative disease and, also, after operative decompression. A number of cases reported in the literature show exceptions to this rule. The author adds two cases treated at Axenfeld's clinic. In the first, an otitic abscess of the brain, the choked disk reached its highest degree four days after the trephining and remained at this stage for about two weeks. A second operation was considered but omitted on account of the improvement of the gen-

eral symptoms. The second case is that of a man, aged 45, who developed serous meningitis a few weeks after a contusion of his skull brought on by a fall; choked disks of both eyes, seen on the day of admission to the hospital a few weeks later, became complicated by considerable retinal hemorrhages during the following week, while, at the same time, the condition improved and the vision and field of vision increased.

This phenomenon is explained by the slow shrinking of the swollen optic nerve and by secondary damage done to its tissues. Central vision could return more promptly because the papillomacular fibers were only slightly or not at all affected.

K. L. STOLL.

BENIGN RETINAL CHANGES. M. ROHNER, Klin. Monatsbl. f. Augenh. 84: 748 (June) 1930.

The author describes a form of phlebitis of the retina caused by infection, or occasionally by an injury in which the changes in the retina are transient and disappear without leaving any trace; the origin is supposed to be tuberculous, as pointed out in two reported cases of young patients. The white patches in the periphery, accompanied by dustlike opacities of the vitreous, had totally disappeared at the time of later examinations. The vision remained normal.

A slight decrease of vision was noticed in two other cases in which the patches remained stationary, although they resembled the phlebitic type; tuberculosis or syphilis was not present.

A fifth case showed a stellated change in the macula which remained stationary for seven years. Glistening white spots had been seen at the first examinations in the surrounding retina; also albumin was found at times in the urine of this girl, aged 7. Her vision, at the beginning reduced to one-third, became normal again.

K. L. STOLL.

CHANGES IN RETINA IN PATIENTS WITH RENAL DISORDERS. K. URWYLER, Schweiz. med. Wchnschr. 60: 489 (May 24) 1930.

Urwyler states that ophthalmologic examinations on 116 patients with renal diseases revealed that in more than one third of the cases there existed changes in the retina. In cases of secondary contracted kidney caused by chronic nephritis, retinal changes were noted in 50 per cent of the cases. The ophthalmoscopic observations did not indicate the nature of the renal disease. However, changes in the retina concurred with arterial hypertension, and they were not connected with the retention of urea in the blood.

[J. A. M. A.]

OPERATIVE TREATMENT OF DETACHMENT OF THE RETINA WITH ELECTRO-ENDOTHERMY AND TREPHINING. SVEN LARSSON, Acta ophth. 8: 172, 1930.

Following favorable results of electro-endothermy (diathermy and electrocoagulation) on detachment of the retina experimentally produced in rabbits, Larsson utilized the method for retinal repair in seven human eyes. He produced an adhesive choroiditis by this electric method.

The detachment is localized, then the conjunctiva is dissected away from the limbus to include the area of detachment. The sclera is carefully freed from blood. The muscles in the field of operation are severed at their attachment, and a suture passed through them. A canthotomy often allows better approach to the eyeball. The conjunctiva is then held away from the field of operation by sutures caught by hemostats. The bleeding in the area is checked by electrocoagulation. The large negative electrode is placed on the leg. A small globular exposed electrode about 2 to 3 mm. in diameter is applied to the sclera. A current which produces the slightest visible spark on the poles in the machine is employed. The positive electrode is applied to many points on the sclera in the area of detachment for an exposure of five seconds. The heat reaction on the sclera manifests itself in the form of an opaque, superficial, dark-colored circular zone. One or two Elliot trephine holes are made in the field of operation to remove the coagulating blood and maintain normal intra-ocular pressure. The conjunctiva and muscle are then reattached. The patient is confined to bed for from eight to fourteen days after the operation. The author procured favorable results in five of seven cases by this method. Mention is made of the simplicity of the method and the unnecessary procedure of search for the hole or rupture which Gonin advocates.

A. M. YUDKIN.

Trachoma

BACTERIUM GRANULOSIS IN RELATION TO TRACHOMA: RECOVERY FROM EXPERIMENTALLY INFECTED MONKEYS AND FROM HUMAN TRACHOMA. E. B. TILDEN and J. R. TYLER, J. Exper. Med. **52:** 617 (Oct.) 1930.

Six additional strains of *Bacterium granulosis* have been isolated by Tilden and Tyler from cases of trachoma occurring in the Indian schools of Arizona. The cultures thus obtained are identical morphologically and culturally with those isolated by Noguchi and have induced the same chronic granular conjunctivitis in monkeys. Several additional cultural and biologic characteristics of *Bacterium granulosis* are described. Of importance is the fact that cocaine, in contradistinction to procaine, has a bactericidal effect on the organism. This fact, in view of the common use of cocaine for anesthesia, may explain the negative results of cultivation experiments reported by some workers.

[J. A. M. A.]

ON THE THERAPY OF TRACHOMA. D. LYRITZAS, Arch. f. Augenh. **102:** 377 (Dec.) 1929.

The author agrees with Kuhnt that in order to obtain a cure it is necessary to treat these cases surgically. This is especially true in countries infested with trachoma. He regards the combined excision of the tarsus as the best procedure. According to his view, local therapy does not suffice since the infection remains deep in the tarsal tissue. Such remaining infection explains the many recurrences which one sees. After removal of the tarsus, the local application of drugs is effective in destroying remaining bacteria.

F. H. ADLER.

Tumors

A Case of Epithelioma of the Meibomian Gland. D. Lazarescu, E. Lazarescu and E. Ionescu, Brit. J. Ophth. **14:** 588 (Nov.) 1930.

The authors refer to the rarity of cases of epithelioma of the meibomian gland. To date they have found forty-eight cases of meibomian cancer reported in the literature. Without histologic examination, the diagnosis in the early stages is practically impossible, but a recurrence of a chalazion after surgical intervention should always make one think of the possibility of cancer. Metastasis is peculiarly apt to occur, the growth invading the orbital cavity and the palpebral conjunctiva.

The case reported was seen in a man, aged 67. The condition began as a small lump the size of a pinhead in the outer half of the ciliary border of the lid. It was circular, clearly subcuticular, and bulged under the conjunctiva. After curettement the growth recurred, and later was removed by means of a V-shaped incision, the resulting gap being sutured.

The microscopic examination showed the tumor near the free edge of the lid, the edge of which was more involved, particularly toward the conjunctival surface. The skin was not invaded. Normal meibomian acini could be traced toward the base of the eyelid; compared with these acini, those of the tumor were from twenty to thirty times larger. The tarsus was compressed, and the muscle layer thinned. The tumor was formed of approximately six lobuli. They were separated one from another by thin fibrous septums which carried blood vessels, which, in places, penetrated into the substance of the growth. The lobules were surrounded by the neighboring conjunctival tissue which, on account of the compression, took on a capsule-like disposition. The cells, which had proliferated and had given rise to the growth, were generally vesicular, concentrically arranged, with clear protoplasm and small nuclei; but there were also cells with scanty protoplasm and larger nuclei, some of which showed kariokinesis. The cells were irregularly placed and crowded together at some points of the growth, and in some places necrosis had occurred, giving rise to vacuolar zones. In the mass of the growth no single meibomian acinus was seen to be normal. The sudan III stain showed well the meibomian acini on one side, and on the other, the Zeis glands situated at the bases of the lash follicles. The tumor was also well stained in sudan III, but here the staining was more diffuse and not so homogeneous as in the normal glandular acini.

W. Zentmayer.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

A. G. FEWELL, M.D., *Secretary*

Oct. 16, 1930

A RÉSUMÉ OF THE SPECIAL INQUIRY ON THE HEALTH OF THE EYES IN PHILADELPHIA

DR. HAVEN EMERSON, New York: After referring to the high standing of Philadelphia as an ophthalmologic and medical center, Dr. Emerson recalled the fact that Philadelphia led in making an extended study of the eye sight of school children as far back as 1878, under the chairmanship of Dr. Samuel D. Risley, and that a survey of lighting conditions in public schools had been conducted actively in 1875. Quoting from an editorial in the *Philadelphia Press* of Sept. 20, 1878, Dr. Emerson pointed out how shrewdly Dr. Risley's survey committee secured the cooperation of the press in preparing the minds of the public for this first study of the vision of school children. The work of Dr. Risley's committee was monumental and served as a guide all over America.

Dr. Emerson then reviewed the general hospital and health survey work of the city, pointing out how nearly every phase of that study had some lesson in it of value in health of the eyes. He featured particularly the social hygiene survey and the notorious abuses of quacks and charlatans in taking advantage of the gullible, pointing out that Philadelphia is really a center for the retail distribution of misinformation conducted on a scale not found exceeded anywhere in America.

Subsequently, Dr. Emerson discussed in considerable detail the lack of complete reports by physicians, hospitals and others of the occurrence of sore eyes in babies. He complimented particularly the work of the Visiting Nurse Society in this as in all other phases of their work. He concluded his discussion with a strong plea that the Ophthalmologic Section of the College should maintain a standing committee to cooperate with the Health League in securing adequate support for the city health department in promoting a much needed extension of its health services in ophthalmology and in improving conditions so obviously needing correction in the public and parochial school systems.

DR. B. FRANKLIN ROYER: Dr. Royer discussed in some detail the incompleteness of the dispensary records in practically every eye clinic in which histories were reviewed. He stated that with the aid of the

ophthalmologists on the committee a sort of measuring stick was designed by using thirteen different conditions which all were agreed would require watchful care in the eye clinic and many of which would be likely to cause deterioration of vision if neglected. The ophthalmologists of the committee estimated the number of return visits that ought to be expected in each condition and the duration of treatment; then, with the aid of a well trained statistician and the complete cooperation of various persons charged with the responsibility for these records in the outpatient departments, 738 records were reviewed and contrasted with the norm established by the committee. It was distressing to find such a tremendous waste of professional effort shown throughout all of these clinics. So large a proportion of the patients attended a totally inadequate length of time that the physician was given no chance to demonstrate what could be done by curative medicine, and a large proportion of the patients were lost to the clinic without showing what result, if any, had been secured. Many patients were found with serious conditions of the eyes, such as interstitial keratitis, keratoconjunctivitis, phlyctenular and choroidal disease or uveitis, with attendance continuing scarcely long enough to establish the diagnosis.

Of the 378 patients reviewed, only 4 were recorded as having made the desirable number of visits to the dispensary.

The point brought out by Dr. Royer was that the efforts of ophthalmologists are largely wasted unless a trained medical social worker serves as the go-between to interpret to the family the scientific lesson which the physician wishes to impress, but in language the patient may readily understand, and to see that those family adjustments are made which will permit return appointments to be kept until such time as the physician has had an opportunity to treat the condition adequately.

The lighting conditions in the public and parochial schools and the adjustment of the shades were discussed in considerable detail, faulty conditions being described and improvements being indicated.

On behalf of the survey committee doing the work, as well as of the National Society for the Prevention of Blindness, Dr. Royer expressed great appreciation to hospitals, dispensaries and other agencies in the city for their splendid cooperation.

MRS. WINIFRED HATHAWAY: Mrs. Hathaway reviewed the survey of the rooms in which sight-saving classes are conducted and of the equipment found in use, and pointed out that through economies in utilizing buildings not available for other purposes some gross faults had been tolerated. She featured the fact, however, that following the survey nearly all of the serious faults had been speedily corrected. She pointed out the need for ophthalmologists to keep in closer touch with the activities, customs and practices of the sight-saving class, both for the purpose of encouraging and guiding the teacher and in order that the practicing oculist might know more about the advantages of the sight-saving class.

DR. L. C. WESSELS (by invitation): Dr. Wessels gave a résumé of the work done in the Eye Clinic at City Hall.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

H. DICKINSON, *Reporter*

Nov. 13, 1930

LESLIE PATON, *President, in the Chair*

VASCULAR TUMORS OF THE BRAIN AND SPINAL CORD (NEUROLOGIC STANDPOINT). DR. A. LINDAU, Sweden.

In the first group of these tumors a systemic disorder can be distinguished: trigeminal nevi, a homolateral pial angioma showing calcification under roentgen examination, and a unilateral buphthalmos or glaucoma, forming together a characteristic syndrome.

The second group consisted of the hemangioblastomas; their favorite site was the hindbrain and spinal cord. They might be either solid or cystic. In obscure cases Perdrau's stain was of great value.

VASCULAR TUMORS OF THE BRAIN AND SPINAL CORD (OCULAR MANIFESTATIONS). MR. E. TREACHER COLLINS.

Congenital ocular hematogenous formations occur in connection with both the retinal and the ciliary vessels. Those of the retinal vessels can be divided into two classes, like those of the brain described by Cushing and Bailey: (1) those composed of fully formed blood vessels, which, Cushing and Bailey said, scarcely deserved to be regarded as tumors, and which they termed "angiomatous formations," and (2) those composed of the cellular elements of blood vessels, which may without equivocation be regarded as neoplastic, and which can appropriately be called "angioblastomas." In the retina the first class presents itself as unusual tortuosity or abnormal branching of fully formed vessels, which remain indefinitely unchanged and unaccompanied by visual disturbance. The second class (the angioblastomas) presents the strikingly characteristic appearance of a raised mass at the periphery of the fundus, composed of rudimentary capillary vessels and endothelial cells to and from which an enormously enlarged companion artery and vein pass; the disease is always progressive, and results ultimately in complete loss of sight.

The first class (those showing tortuosity of the retinal vessels) may occur without angiomatous formations elsewhere, or in association with angiomas of the skin and mucous membranes, and sometimes also with epileptic or other symptoms suggesting the presence of an angiomatous mass pressing on the cerebrum. The second class, the angioblastomas, can be associated with cystic angiomatous growths in the cerebellum, as Dr. Lindau first pointed out. Just as the recognition of this condition in the eye aids in the preoperative diagnosis of disease of the cerebellum, so the recognition of the presence of tortuosity of the retinal vessels may aid in the preoperative diagnosis in a case exhibiting symptoms of pressure on the cerebrum.

Tortuosity of the retinal blood vessels is sometimes associated with recurrent attacks of epistaxis, owing to the presence of vascular nevi of the nasal mucous membrane. An angiomatous condition of the

retinal blood vessels may account for the recurrent attacks of intra-ocular hemorrhage met with in young men in association with recurrent nosebleed, a condition first described by Eales, who noted that in all his cases "the retinal veins at the periphery were found to present remarkable corkscrew-like tortuosities."

The retinal or cerebellar angioblastomas are neoblasts of mesoblastic tissue growing in an epiblastic structure. In association with the rudimentary capillary formation some proliferation of the perivascular endothelium is often met with, the cells being loaded with globules of fat. In rare cases the endothelial new growth preponderates over the vascular formation, a hemangio-endothelioma being produced.

The characteristic ophthalmoscopic appearance of an angioblastoma of the retina is due to the fact that the retinal arteries are end-arteries. Hemorrhage from these growths frequently occurs, the blood collecting in the substance of the retina, in the vitreous chamber, or between the retina and choroid. Fibrous tissue replacing blood-clots in the vitreous, or occurring between the retina and choroid, gives rise to "retinitis proliferans" and "massive exudation," often observed in connection with these growths.

The angiomas encountered in the choroid are usually of the cavernous type, and in eyes so affected, detachment of the retina and glaucoma nearly always ensue. They are associated with vascular nevi of the skin of the face, and in many of the recorded cases the inner surface of the growth in the choroid has been found encapsulated by a fibro-osteogenetic membrane. In intracranial angiomas situated above the tentorium, Weber and others found roentgenographically that the surface of the growth is bounded by a membrane giving a dark shadow, which is probably osteogenetic like that found in the eye.

The anterior ciliary blood vessels on the surface of the eyeball sometimes participate in an angiomatous formation involving the skin of the face. More frequently some disturbance occurs in the circulation of the intra-ocular fluid, resulting in increased tension of the eye, and in young children in buphthalmos. Cushing recorded three cases of spontaneous hemorrhage with trigeminal nevi and buphthalmos on one side, and contralateral epileptic symptoms. Weber collected eight cases of unilateral buphthalmos with cutaneous and cerebral, not cerebellar, hemangiomas.

(The discussion was mainly on neurologic lines.)

Book Reviews

SUCCÈS OPÉRATOIRES DANS LE TRAITEMENT DU DÉCOLLEMENT RETINIEN; EST-IL INDISPENSABLE D'OBTURER LA DÉCHIRURE? By GABRIEL P. SOURDILLE. Paper, 18 francs. Pp. 112. Paris: Amédée Legrand, 1930.

The author took as subject for his thesis the work done by his father, Prof. Gilbert Sourdille of Nantes, on the treatment for retinal detachments which the son had the opportunity of following up in all its details. After passing in review in the first two chapters the theories on the pathogenesis and the numerous methods of treatment, he gives a detailed description of the Sourdille method.

The principle underlying Sourdille's procedure is as follows: On account of a pathologic process going on in the vascular system of the choroid and principally in the choriocapillaris, a separation takes place between the retina and its pigment epithelium layer, and fluid accumulates underneath the retina. The object of the treatment is to evacuate this fluid as completely as possible and to cause a reaction in the choroid which should result in an adhesion between it and the retina. The choroidal reaction should not be too great or insufficient, and the main difficulty is in gaging exactly the intensity of this reaction. To obtain a reattachment of the retina, Sourdille uses the cautery in some cases, retinal punctures with the Graefe knife in most of the cases and both means at the same time in others. He advises the use of retinal punctures with the knife in detachments of high elevation (blebs, balloons, etc.), while he limits the indication for the use of ignipuncture only to those cases with flat detachments. He does not believe that cauterization of the margins of the retinal tear is essential, and his results seem to sustain his contention. The essential points of Sourdille's method are: (1) multiple scleroretinal punctures (with knife or galvanocautery), followed by (2) subconjunctival injections of mercury oxycyanide solution directly after the punctures, and (3) complete immobility of the head in such a position as to facilitate the apposition of the retina to the underlying choroid. The details of the technic are: (1) a few days of absolute rest in bed, during which time calcium chlorides are given to prevent choroidal hemorrhage, and (2) scleroretinal punctures performed like any ordinary posterior sclerotomy. The number of punctures depends on the extent of the detachment. In total detachments the author performs four punctures placed midway between the four recti muscles and from 15 to 18 mm. behind the limbus corneae. (3) After the termination of the operation, he injects a few minims of a 1:1,000 solution of mercury oxycyanide under the conjunctiva in the punctured areas. (4) An ointment containing atropine and morphine is then administered in the conjunctival sac, and a binocular bandage applied. The author insists on a rest of from fifteen to twenty-one days.

It is often necessary to repeat the treatment several times. The most valuable part of this thesis is the record of the 79 cases in which successful operations were performed since 1920. The author has treated 170 patients, and his results compare favorably with those published by Gonin. Nineteen of Sourdille's detachments were of more

than three months' duration, and 9 of more than six months. In some of Sourdille's successful cases the patients had been under his observation for from eight to ten years.

Conceding that his patients have to stay perfectly quiet in bed at least one or two weeks longer than those operated on by the Gonin method, Sourdille believes that his procedure presents a number of advantages: the application of the treatment in cases without tears, the avoidance of extensive injury to the retina and choroid and the attempt of anchoring the retina in several places at the same sitting. He also insists on the general treatment of the patient with the object of improving the general circulation and renal elimination, an important suggestion not sufficiently insisted on by the Lausanne surgeon.

The thesis is an interesting piece of work. There is no doubt that in a series of 170 cases complications, accidents and recurrences must have occurred. These are not mentioned in the book.

Perhaps the feeling that there is need of a great deal of optimism on the part of those trying to master the technic and indications for the newer methods for the treatment for retinal detachment has prevented the author from publishing the shady side of the problem. However, science always demands full information.

MARK J. SCHOENBERG.

LEHRBUCH UND ATLAS DER SPALTLAMPEN MIKROSKOPIE DES LEBENDES AUGES. By PROFESSOR ALFRED VOGT, Zurich. Second edition. Part 1. Price, 178 marks. Pp. 313, with 692 illustrations, mostly in colors. Berlin: Julius Springer, 1930.

The first edition of the author's well known Atlas of Slit-Lamp Microscopy, which appeared in 1921, was rapidly exhausted. So much has been added to this science in the last nine years that Professor Vogt has now enlarged the original into the present form of textbook as well as atlas and has added many new illustrations. The author believes that the importance of the principle of the "optical section" is not as generally shared as it should be, and that the time is not far distant when the slit-lamp will be just as much a part of the oculist's armamentarium as is the ophthalmoscope. The present work is to be divided into the three following volumes: 1. Introduction; Methods of Examination and Technic. Cornea and Anterior Chamber. 2. Lens and Zonule. 3. Iris, Vitreous, Conjunctiva and Lids, and a chapter on optical illusions and phantoms.

The present volume (no. 1) begins with a description of the slit-lamp, the methods of examination and the particular technic required. Part 1 deals with the normal cornea and the normal limbus, then follow the senile cornea, corneal degenerations, ocular changes in pseudosclerosis, inflammatory changes in the cornea and their sequelae, injuries of the cornea and their sequelae, nontraumatic ruptures in Descemet's membrane, glass ledges at the posterior corneal surface, erythrocyte columns on the posterior cornea, obscure corneal changes and pathologic and senile changes at the limbus.

Part 2 takes up the anterior chamber under normal and pathologic conditions. A bibliography and index conclude the volume.

Every one who has used the slit-lamp is soon fascinated by the introduction which it gives to the histology of the living eye. Furthermore,

it has advanced knowledge of pathologic processes, and one can look forward to many more contributions in the field in which fixing and hardening methods are so unsatisfactory. Much of the knowledge in this subject has been advanced by Professor Vogt; ophthalmologists owe him a great debt of gratitude that in his busy day he has found time to write this monumental work. The descriptions of the methods of examination are clear and most instructive. In the clinical part, one marvels at the many conditions described. A number of new pathologic conceptions are presented, which are of help in the understanding of these diseases. The beauty of the many colored illustrations is striking and adds to the importance of this remarkable work, which should occupy a prominent place in every ophthalmologist's library. A. K.

DAS GLAUKOM. Handbuch der gesamten Augenheilkunde. By PROF. A. PETERS. Third edition. Linen. Price, 39.80 marks. Pp. 361, with 35 illustrations. Berlin: Julius Springer, 1930.

This is a new edition of the treatise on glaucoma written by Schmidt-Rimpler, in 1908, for the second edition of the Graefe-Saemisch Handbuch. The author has constructed a complete study, with a bibliography from January, 1908, to January, 1930. The subdivision of glaucoma is the usual one. Under acute glaucoma the subjective symptoms are first described, in which are included the disturbances of the light and color senses, the objective symptoms and differential diagnosis; a description of its different forms follows. In glaucoma simplex the visual field and its variations are first described, then the excavation of the optic nerve head and the differential diagnosis. A chapter is devoted to hydrophthalmos, megalocornea and cutaneous and vascular changes in hydrophthalmos. Absolute and degenerative glaucoma and secondary glaucoma occupy the next two chapters. The remaining chapters deal with the etiology of glaucoma, intra-ocular pressure, the intra-ocular circulation, experimental glaucoma, theories of glaucoma, prognosis and treatment, nonoperative and operative.

The author has succeeded in covering this extensive field in an admirable manner, and the study of the book will be a necessary help to any one undertaking the investigation of the many still obscure problems of this elusive subject. A. K.

MOYENS DE PROTECTION DES YEUX EXPOSÉS AUX RADIATIONS ULTRAVIOLETTES. By DOCTEUR ANDRÉ HALPHEN. Paper, 10 francs. Pp. 76. Paris: Amédée Legrand, 1930.

It has long been established that the short wave radiations of the spectrum, particularly the ultraviolet rays, have a deleterious effect on the anterior segment of the eye. Protection by means of screens and glasses is sufficient in most cases, but moving picture actors, by the very nature of their calling, are denied this relief. The author undertook to discover a method to protect these people by some means that would remain invisible.

After a series of experiments on rabbits, he discovered that ethyl diphenylmaleate absorbs ultraviolet rays up to 3,000 angstrom units. When dissolved in castor oil in the proportion of 2.9 per cent and instilled into the eye at intervals of ten minutes, it is well tolerated and renders perfect protection. G. M. BRUCE.

Directory of Ophthalmologic Societies *

FOREIGN

Oxford Ophthalmological Congress

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6, Avenue de La Motte Picquet, Paris 7è.
Place: Paris. Time: Second week of May, 1931.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. George F. Suker, 25 E. Washington St., Chicago.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Asheville, N. C. Time: June 3, 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana. Time: Sept. 14-19, 1931.

American Ophthalmological Society

President: Dr. Arnold Knapp, 10 E. 54th St., New York.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: Asheville, N. C. Time: June 3, 1931.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Donald H. O'Rourke, 217 Imperial Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts, Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Hendrie W. Grant, Lowry Bldg., St. Paul.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, 1005 Medical Arts Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.
President: Dr. Fletcher D. Woodward, Box 162, University.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. S. S. Hall, Fairmont.
Secretary: Dr. G. A. Smith, Montgomery.
Place: Clarksburg: Time: May 19-21, 1931.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. McDougall, Atlantic National Bank Bldg., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: Dr. William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. George B. Potter, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 425 Aquila Court, Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. T. J. Walthall, 728 Main Ave., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. F. Swett, 693 Sutter St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.